PLANT PATHOLOGY

A RECORD OF CURRENT WORK ON PLANT DISEASES AND PESTS

Published quarterly for the
Ministry of Agriculture, Fisheries and Food
Plant Pathology Laboratory
Harpenden, Herts

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FIELD TRIALS OF DDT AGAINST FRIT FLY ON SPRING OATS

by D. W. EMPSON

National Agricultural Advisory Service, Wolverhampton

BY 1951 the difficulty of growing satisfactory spring oats in the West Midlands led many farmers, especially in the Golden Valley area of Herefordshire, to omit this crop from of the rotation. A survey in 1952 suggested that frit fly might be one of the main factors involved. To test this, and at the same time to investigate the effectiveness of DDT spraying, four field trials were arranged for 1953. To reduce reinfestation by the pest the plots were as large as possible, there being no replication. Results from these pilot trials were promising enough for the N.A.A.S. Field Experiments Committee to sponsor a series of similar trials in all counties in the Province. In 1954 I became responsible for the entomological side and for the analysis of results.

Fields of late-sown oats were selected, so far as possible, on farms where both a crop sprayer and a combine harvester were available. Each field was divided into two or more areas of 3–4 acres; one of these areas was selected at random for the "sprayed" plot, the remainder of the field being the "control". All fields were over 6 acres, the majority being between 8 and 12 acres.

The treatment consisted of two spray applications, each of 3 pints of 20 per cent miscible DDT in 30–40 gallons of water per acre. The first spray was to be applied during the first week of May or during the one-leaf stage of growth, whichever was the later. The second was to be fourteen days after the first. In practice it was found that weather conditions often caused delay: on average the first spray was delayed by about five or six days and the mean interval between spray applications was eighteen days.

In 1954 and 1956, counts of attacked and healthy shoots were made during June on some of the fields. Where possible, yields of the sprayed and control plots were estimated, and an estimate was also made of the degree of frit fly grain attack on each plot.

TABLE 1

Number of Trials in each Year

Trials	1954	1955	1956	Total
Set up and sprayed	25	15	9	49
Counted at tiller stage	17	0	7	24
Grain attack estimated	19	14	6	39
Yield estimated	12	15	7	34

Table 1 shows the number of trials started and completed in each year. Failures to obtain final yields were mostly due to bad harvesting conditions. During the series two trials were discarded because of severe cereal root eelworm attack but all other fields were included in the final result provided satisfactory yield assessment was possible. In 1954, seven fields were too badly laid for accurate

harvesting, but were sampled for the estimation of frit fly grain attack. There were eleven varieties represented in the trials but Blenda, Sun II and Star accounted for over half the crops.

FRIT FLY ATTACK

Estimates of the intensity of shoot attack in June were made by counting the total, and visibly attacked, shoots in ten sample lengths of row per plot. The sample unit was two adjacent one-foot lengths of row, the counts being made at approximately equal intervals along one diagonal on each plot.

TABLE 2

Mean Counts of Total and Attacked Shoots per 20 ft of Row

Year	Shoots:	Sprayed	Control	Difference	Degrees of Freedom	Significant at p =
1954	Total	435	447	12±12·3	16	NS
	Attacked	16	33	17± 4·3	16	0·01
1956	Total	556	590	34±19·5	6	NS
	Attacked	35	109	74±17·4	6	0·01
Both Years	Total	470	489	19±10·4	23	NS
	Attacked	21	55	34± 7·7	23	0·001

There was no significant difference in the total number of shoots, but spraying with DDT caused a reduction of about 60 per cent in the number attacked by frit fly.

Small samples were taken at harvest to estimate frit fly grain attack. From each plot 150 "grab" sub-samples of three or four panicles were taken at random and bulked to form the final sample. This was threshed by hand and from it were taken at random, by "coning and quartering", 1000 main grains and 1000 bosom grains. The number of main grains in 1000 random grains was also determined and used as a correction to give an overall estimate of grain attack.

TABLE 3

Mean Percentage Grain Attack (in Angular Transformation)

Year	Sprayed	Control	Difference	Degrees of Freedom	Significant at p =
1954	9·4	10·6	1·2±0·61	18	NS
1955	16·3	18·7	2·4±0·66	13	0·01
1956	20·4	26·0	5·6±1·99	5	0·05
1954–56	13·6	15·9	2·3±0·53	38	0·001

Table 3 gives the analysis (in angular transformation) of the data from the "grab" samples. The overall difference, although small, is significant. In at least two of the three years spraying in May produced a real reduction in grain attack, and this reduction appears to have been greatest in the year (1956) of heaviest attack. The mean percentage attacks for the three years together (de-transformed from angles) were: control plots 7·6, sprayed plots 5·4.

YIELD

Yields of the sprayed and control plots were estimated by averaging the yields from four combine harvester "cuts", each of at least 60 yards length from each plot. In a few instances yields were taken from separate threshing of the entire produce of each plot.

TABLE 4

Mean Yields (cwt per acre at 14 per cent Moisture Content)

Year	Sprayed	Control	Difference	Degrees of Freedom	Significant at p =
1954	26·2	24·0	2·2±1·02	11	NS
1955	23·5	22·0	1·5±0·61	14	0·05
1956	17·6	13·0	4·6±0·51	6	0·001
1954–56	23·3	20·9	2·4±0·49	33	0·001

The analysis of the mean yields from the thirty-four completed trials is given in Table 4. In at least two out of the three years DDT spraying gave an increase in yield; the greatest increase was in 1956, when frit fly attack was severe (Tables 2 and 3) and yields were low (Table 4).

TRIALS IN OTHER PROVINCES

Trials similar to those reported above were carried out in 1954 and 1955 by the advisory entomologists in the South Eastern (Reading), South Eastern (Wye), East Midland and Northern Provinces. The results are summarized below.

In the Reading area in 1954 two trials with DDT (applied twice as in the West Midland trials), and two with parathion (applied once at 1 pint of 20 per cent miscible in 10 gallons of water per acre) indicated that both treatments gave a reduction of about two-thirds in the numbers of shoots attacked by frit fly. The gain in yield was estimated to average about 4 cwt per acre. Counts of attacked shoots from different parts of the sprayed areas showed that considerable reinfestation took place from around the headlands and from the unsprayed areas. There was an indication that reinfestation was greater from the edges of the fields than from the control plots. In 1955 frit fly attack was slight, but from five trials it appeared that DDT caused a greater reduction of attacked shoots than did parathion. Dieldrin, however (two applications of 2 pints of 15 per cent miscible in 30 gallons of water per acre) may have been rather more effective than DDT. One trial, in which the single spray of parathion was applied on three different dates, indicated that the correct timing of the application is important; and this may account for variable results with single sprays of this insecticide.

There were two trials, both with DDT, in the Wye area in 1954. Counts of attacked shoots were not made but the gains in yield were estimated to be 3·4 and 5·0 cwt per acre.

A similar trial with DDT in the East Midland Province in 1954 gave a reduction of 50 per cent in the number of attacked shoots and a gain in yield of 2·4 cwt per acre. In 1955 in the same Province there were three trials with DDT but estimates of yield were not obtained. Only in one of these crops was an appreciable attack by frit fly encountered and here the DDT was not very effective. The shoot attack was 25·8 per cent on the control area and 17·8 per cent on the sprayed area.

Four trials in the Northern Province in 1955 differed from the other DDT trials in that only one application was made (3 pints of 15 per cent DDT emulsion in 25 gallons of water per acre). This was given when the adult flies were becoming active as judged by direct observation. The mean gain in yield from this treatment was 1·9 cwt per acre.

There was no significant difference between the mean percentage grain attacks in the eight trials for which counts are available. It is of interest to note that none of the crops in the Northern Province was attacked at the grain stage. This agrees with the low counts of attacked grains from this area found in the 1947–51 survey (Thomas, 1954).

Of the eighteen trials in other Provinces yield estimates are available for nine, in eight of which DDT was used and in one, parathion. The control mean yield of 24·9 cwt per acre was increased by $3{\cdot}0 \pm 0{\cdot}48$ (8 df.) to 27·9 cwt per acre on the treated plots.

DISCUSSION

The reduction by 60 per cent in shoot attack may be sufficient to prevent loss from this source. Each oat plant may produce a number of tillers but in a normal healthy commercial crop only the main shoot contributes to yield. Where the main shoot is damaged one or more tillers will form heads, but there will be a reduction of yield (Cunliffe, 1925; Tincker and Jones, 1931). If the shoots protected by DDT are the main shoots then the attack would be confined to non-yielding tillers, and would probably have little effect on the final crop.

Flies produced from these tillers, however, give rise to larvae in the grain and this may explain the failure to achieve more than a slight reduction in grain attack. It is also possible that the plots were not large enough to produce much reduction in reinfestation from the control areas and indeed reinfestation may arise mainly from sources outside the crop.

The mean gain in yield of 2·4 cwt per acre was only marginally profitable and on some crops, even though sown late, spraying would not give an economic return. On the other hand many crops have shown benefits of 6 cwt per acre or more, and there was no case of a heavy frit fly attack where spraying did not produce a profitable increase in yield.

The data are not sufficient to permit of comparisons of gain from spraying in different counties or between crops sown at different times. No critical observations have been made on the influence of DDT on the other fauna of oat fields; only immediately after spraying were any differences in insect activity apparent.

The work was under the direction of Mr. H. C. F. Newton. The selection of sites, spraying and harvesting were carried out by members of the county staffs of the West Midland Province, N.A.A.S. to whom thanks are due. Grateful acknowledgement is made to the many farmers who provided facilities for this work, usually at a particularly busy time of year. Thanks are also due to the advisory entomologists concerned for permission to include results of trials carried out in other N.A.A.S. Provinces.

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ERGOT INFECTION OF GRASSES IN THE SOUTH-WEST OF ENGLAND

by J. G. JENKINSON

Seale-Hayne Agricultural College, Newton Abbot, Devon

GARNER (1957) reports that losses in livestock from ergot poisoning (due to infected grass) are relatively frequent. In the South-west, extreme forms of the disease are uncommon, but Fincham (1957 *in lit.*) has reported the occurrence of the diarrhoeic form in cattle which cleared, or recurred, with movement on and off infected pastures. In view of the dangers which may result from stock grazing infected pastures investigations were carried out at Seale-Hayne in 1956 and 1957 to obtain further information on the extent and conditions of infection.

HOST RANGE AND INCIDENCE

Ergot infection of grasses is of common occurrence in the South-west. *Lolium perenne* and *Lolium multiflorum* are the species most frequently attacked, although infection is common on *Festuca arundinacea, Festuca pratensis, Holcus lanatus* and *Agropyron repens*. Ergots have also been collected from *Agropyron pungens, Agropyron caninum, Dactylis glomerata, Agrostis stolonifera, Phleum pratense, Poa pratensis, Poa trivialis, Poa annua, Alopecurus pratensis, Arrhenatherum elatius, Molinia caerulea, Elymus arenarius, Bromus erectus, Sesleria caerulea, Ammophila arenaria, Hordeum murinum, Glyceria fluitans, Nardus stricta* and *Lagurus ovatus*. In these records no attempt has been made to differentiate between infections caused by *Claviceps purpurea* (Fr.) Tul. and *C. microcephela* (Wallr.) Tul. No ergots have been found on either *Triticum aestivum* or *Secale cereale*, although Mastenbroek and Oort (1941) report that some strains of *Claviceps purpurea* which infect ryegrass can also infect rye. A survey carried out on the Seale-Hayne College farm in September 1957 showed that the percentage of infected ryegrass inflorescences in different fields ranged from 2 to 88 per cent, and that the average number of ergots per infected inflorescence increased with the percentage of infected inflorescences, and varied from 1 to 3·8. The lowest figures were obtained from fields which had been cut three or four times during the season, and the highest from grassland that had never been mown.

GERMINATION OF ERGOTS, AND ASCOSPORE DISCHARGE

The date of germination was determined in 1956 for ergots which were harvested and placed outside in pots containing vermiculite, and also for those which occurred naturally in the field. Ergots were considered to have germinated when the stromata first became visible. In the pot experiments, the percentages of ergots from perennial ryegrass that had germinated by various dates were as follows:

Date	June	1	6	11	16	21	27
Percentage germinated		0	37	60	76	89	95

The first signs of germination are the development of small longitudinal cracks from which the stromata are eventually developed. A period of about seven days normally elapses between the first appearance of the stromata and the development of mature ascospores. Ascopore discharge from individual ergots may occur at intervals for a period of about fourteen days. No significant differences were observed in the germination date of ergots gathered from other grasses.

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In the field, germination of ergots was observed over the period June 6 to July 20 in 1956. Observations in 1955 and 1957 confirm that ergot germination normally occurs about the first week in June, but the exact time is dependent on rainfall, as the ergots have to be moist for several days before germination occurs.

DEVELOPMENT OF ERGOT INFECTION IN THE FIELD

To measure the rate of increase in the percentage of infected inflorescences in the field, recordings were made in 1956 and 1957 on a series of plots of perennial ryegrass which were in flower at intervals from the end of May to the beginning of August. The variation in the flowering dates of the different plots was achieved by using an early strain (Irish Commercial) and a late strain (S.23) of ryegrass, and by varying the time of cutting. Each treatment was replicated six times each year. The size of each plot, 1 yd × 1 yd, was sufficiently small to enable all the ryegrass inflorescences to be examined individually, and for the number infected with either the conidial or the sclerotial stage of the fungus to be recorded. Recording of the inflorescences was normally delayed until most of them were mature, and this allowed practically all the infections to develop into the sclerotial stage. The results obtained in the two years are given in the Table.

Relationship Between Time of Flowering and Ergot Infection in Perennial Ryegrass, 1956 and 1957

Treatment *Strain and date of cutting*	Flowering Period	Inflorescences		
		Mean total No. per plot	*Mean percentage infected*	*Range of percentage infected in six replicates*
1956				
Irish Commercial, uncut	June 5 to June 20	2,313	0·01	0— 0·04
Irish Commercial, cut March 17	June 10 to June 30	2,121	0·09	0— 0·17
S. 23. Uncut	June 20 to July 24	1,133	4·56	2·66— 9·09
Irish Commercial, cut March 17 and June 9	July 1 to Aug. 2	464	22·68	15·16—22·40
1957				
Irish Commercial, uncut	May 25 to June 10	1,664	0·10	0·08— 0·12
Irish Commercial, cut March 20	May 28 to June 21	995	0·18	0·09— 0·32
S. 23. Uncut	June 23 to July 27	308	5·90	4·31— 7·17
Irish Commercial, cut May 29	July 1 to July 28	56	15·47	11·76—20·00
S. 23. Cut June 10 ..	July 7 to Aug. 13	63	19·38	14·03—25·00

Irish Commercial and S.23 ryegrasses appeared to be equally susceptible to ergot infection. Plotting the dates of maximum anthesis for each treatment, obtained by eye estimates of the plots, against the percentage of infected inflorescences (Fig. 1) shows that the rapid increase in the numbers of infected inflorescences occurred approximately fourteen days earlier in 1957 than in 1956. The difference is probably due to the heavier rainfall in May 1957 which initiated ergot germination about three weeks earlier than in 1956. It is of interest that the ryegrass also came into flower earlier in 1957 than in 1956, and that consequently early flowering coincided with early germination of the ergots.

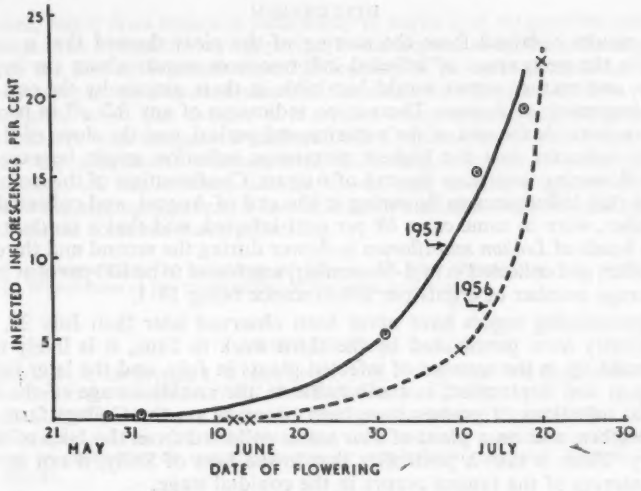

Fig. 1. Relationship between date of flowering and percentage of ergot infected ryegrass inflorescences, 1956 and 1957.

In 1957, the total numbers of ergots in the six one-yard-square plots per treatment were recorded. In Fig. 2 these numbers are plotted against the harvesting dates for each treatment. Treatments were harvested as soon as all the ergots were fully developed.

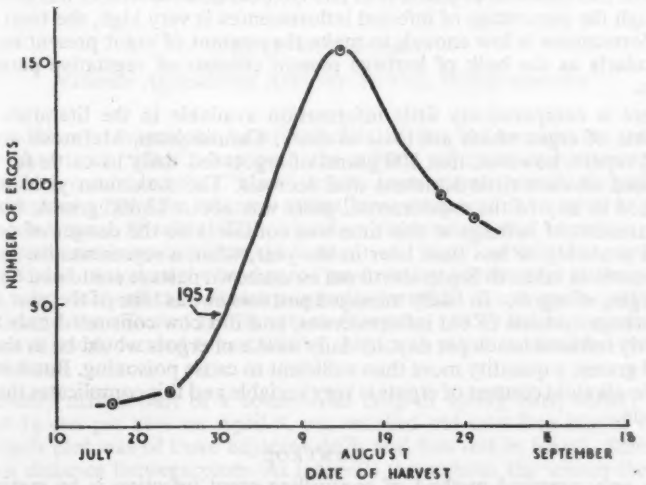

Fig. 2. Relationship between date of harvest and total number of ergots produced in the six one-yard-square plots for each treatment in 1957.

DISCUSSION

The results obtained from the scoring of the plots showed that a rapid increase in the percentage of infected inflorescences occurs about the beginning of July, and mature ergots would be visible in these grasses by the end of July or the beginning of August. There is no indication of any fall off in percentage infection towards the end of the experimental period, and the slope of the curve (Fig. 1) indicates that the highest percentage infection might be expected in grasses flowering later than the end of August. Confirmation of this comes from the fact that inflorescences flowering at the end of August, and collected in late September, were in some cases 88 per cent infected, and that a random sample of fifty heads of *Lolium multiflorum* in flower during the second and third weeks in October, and collected in mid-November, was found to be 100 per cent infected, the average number of ergots per inflorescence being 14·1.

As germinating ergots have never been observed later than July 20, and as the majority have germinated by the third week in June, it is likely that the rapid build-up in the number of infected plants in July, and the later infections in August and September, is attributable to the conidial stage of the fungus. Conidial infections of grasses have been observed on the College farm as late as December, and on a plant of *Poa annua* collected from the Isles of Scilly in January. There is thus a possibility that in the Isles of Scilly, if not in Devon, overwintering of the fungus occurs in the conidial stage.

Although the percentage of infected inflorescences increases until very late in the year, the decrease in the total number of inflorescences means that the number of ergots produced per acre (Fig. 2) reaches a maximum about mid-August and then falls off quite quickly. This does not mean that the danger period for grazing animals is confined to this month, as the important factor is the proportion of ergots by weight to the total weight of herbage available; the total amount of herbage falls in September and October, thus increasing the length of the danger period. The greatest danger probably occurs in pastures which have been grazed during the summer and where, in September, much of the herbage consists of mature inflorescences, previously neglected by grazing animals. Examination of pastures in late October and November has shown that, although the percentage of infected inflorescences is very high, the total number of inflorescences is low enough to make the amount of ergot present negligible, particularly as the bulk of herbage present consists of vegetative parts of the plants.

There is comparatively little information available in the literature on the amounts of ergot which are toxic to stock. Cunningham, McIntosh and Swan (1944) report, however, that 100 grams of ergots fed daily to cattle for 14 days produced characteristic lameness and necrosis. The maximum yield of ergots obtained in any of the experimental plots was about 12,600 grams, but as the total amount of herbage at this time was considerable the danger of poisoning would probably be less than later in the year, when a representative sample of inflorescences taken in September from an unmown pasture contained 6 per cent, by weight, of ergots. In badly managed pastures at this time of the year much of the herbage consists of old inflorescences, and if a cow consumed only 10 lb of similarly infected heads per day, its daily intake of ergots would be in the region of 280 grams, a quantity more than sufficient to cause poisoning. But it is known that the alkaloid content of ergots is very variable and this complicates the dosage question.

CONTROL

The only practical method of controlling ergot infection is by cutting after grazing. It is of interest that the survey carried out on the College farm showed that even repeated cutting throughout the year does not entirely eliminate ergot

in all cases, but it does reduce it sufficiently to make it of no possible economic importance. The critical flowering period would appear to be from the beginning of July to the end of September, and if topping of pastures grazed during this period was adopted as a routine measure, the incidence of ergot would be reduced to a negligible level. The danger of a serious ergot infection developing is, of course, greatest with ryegrasses such as Irish Commercial which flowers abundantly in the aftermath; whereas with S.23, which normally only produces large numbers of heads when first in flower and smaller numbers of inflorescences in the aftermath, there is less chance of sufficient ergoted inflorescences being produced to cause stock poisoning.

I wish to acknowledge the assistance of Mr. K. C. Vear throughout this work, and of Miss V. B. Whitehouse in the preparation of the table and graphs.

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"BLAST" PRODUCED BY THE ARTIFICIAL DEFOLIATION OF OATS

by D. W. EMPSON

National Agricultural Advisory Service, Wolverhampton

THE condition in which the basal spikelets of the oat panicle are withered, usually known as "blast", has been the subject of much speculation. Some workers have concluded that "blast" results from insect (usually thrips) injury to the developing panicle, whereas others have attributed it to adverse physiological conditions. Sheals (1950), who has summarized the literature on the subject, was unable to correlate the amount of "blast" with insect prevalence, and concluded that it might be due to "an inability on the part of the plant to nourish adequately all the spikelet primordia formed". Work on the oat panicle in relation to the effect of frit fly attack (Empson, 1957) led to a similar conclusion and it was decided to test this by subjecting plants to a sudden reduction of leaf area.

An evenly drilled part of a commercial crop of spring oats, variety Sun II sown at 1½ cwt per acre on April 9, was marked out into four blocks of eight plots. Each plot was of three adjacent drills and two feet in length, there being one foot distance between plots. At intervals throughout the season the plants of one plot in each block were defoliated by hand, every leaf being cut about ½ inch above the ligule. Thus all plants, except the controls, were cut once during the season.

At harvest (August 16) the centre one-foot length of the middle row of each plot was carefully dug up and taken to the laboratory for drying and examination. Unfortunately some samples from the fourth block were damaged by mice; this block was therefore discarded leaving three replicates of each treatment.

The dates of defoliation and the respective growth stages are given in Table 1. For the early stages ten plants were taken on each occasion from between the plots, measured from the lowest stem node to the tip of the longest leaf and (after storage in formalin) dissected for examination of the developing panicle. The growth stages are numbered according to the Feekes scale as modified by Large (1954).

TABLE 1

Growth Stages at which Plants were Defoliated, and Consequent "Blast"

Date of Defoliation	No. of Days from Sowing	Growth Stage at Defoliation	Assessment of "Blast" on July 9
May 8	29	Stage 1. Two leaves; mean plant length 5·9 in. A few tillers just visible, panicle primordia just forming (Plate II, 2).	Slight
May 15	36	Stage 2. Three leaves; mean plant length 7·5 in. Most plants with first tiller about 1 in. in length. Panicle primordia showing little advance on Stage 1.	Slight
May 22	43	Stage 3. Four leaves; mean plant length 9·6 in. Most plants with two tillers visible. Panicle primordia becoming differentiated (Plate II, 2).	Slight
June 4	56	Stage 6. Five or six leaves; mean plant length 15·6 in. Immature panicles 4 to 7 mm, mostly with glumes of apical spikelet fully formed (Plate II, 2).	Moderate
June 19	71	Stage 10. Panicle half way up sheath.	Severe
June 26	78	Stage 10.1. Apex of panicle emerging from sheath.	Severe
July 3	85	Stage 10.5. Panicle fully emerged on all except a few plants.	Moderate
Aug. 16	129	Stage 11.3. Crop harvested.	

The last defoliation was carried out on July 3. On July 9, when all the panicles were fully emerged, it was apparent that severe "blast" had been induced in plants defoliated on June 19 and June 26. All plots were assessed according to the amount of "blast" present, the intention being to count the affected spikelets after harvest. It was found that the withered spikelets had blown away by that time so no count was possible. The assessments are given in Table 1 and a typical panicle from the plot cut on June 19 is compared with a control panicle of equivalent size in Plate 2, 1.

The mean numbers of fully formed spikelets per one foot of row are given for each treatment in Table 2. It will be seen that there is a steady rise in this number with the lateness of defoliation except that counts for June 19 and June 26 are approximately 100 spikelets below the general trend. This drop is presumably the result of "blast".

There were no significant differences in the numbers of plants per plot in each treatment nor in the numbers of panicles produced by each plant (mean 1·03). The mean number of whorls per panicle was 6·5 and there was an indication

that this may have been reduced in plants defoliated in mid and late (but not early) May. There were no significant differences between treatments in the proportions of spikelets with one, two or three grains.

TABLE 2

Number of Spikelets and Grain Weights in Relation to Date of Defoliation

Date of Defoliation	Mean No. of Spikelets per Plot	Grain wt (mg)		Percentage Kernel		Yield per Plot g
		Main Grains	*Bosom Grains*	*Main Grains*	*Bosom Grains*	
May 8 ..	328**	42·3	26·0	74·6	80·4	16·89**
May 15 ..	354**	42·0	24·4	75·3	80·4	18·49*
May 22 ..	359*	42·4	24·4	74·8	80·3	17·68*
June 4 ..	369*	34·8**	19·3**	74·4	79·0	15·01**
June 19 ..	273**	38·5	22·7	77·9	81·8	14·17**
June 26 ..	371*	35·5**	19·6**	74·4	78·5	19·82
July 3 ..	463	33·2**	18·6**	72·8*	77·0*	18·43*
Control ..	468	42·5	24·5	75·4	79·6	25·65
S.E. (14 df.)	±30·35	±1·506	±1·272	±0·694	±0·839	±2·206

* Significantly different from control at P=0·05 ; **, at P=0·01

Grain weight (Table 2) was not affected by defoliation in May, but there was a significant reduction in mean weight of both main and bosom grains for plots cut in June and early July, with the exception of that cut on June 19. There appeared to be a tendency for the kernel content (Table 2, Percentage Kernel) to be low on plots with low mean grain weight. Plants cut on June 19 produced grain of very high kernel content, the ratio of kernel to whole grain being well above that normally found in Sun II.

The total yield per plot did not differ significantly between the different dates of defoliation. The uncut control plots yielded more than the others with the possible exception of that defoliated on June 26.

CONCLUSIONS

Typical "blast" in oats was produced by the defoliation of plants shortly before the emergence of the panicle. This indicates that "blast" is a symptom of lowered vitality at a critical time and might be expected to arise from a number of causes; thus we could expect "blast" to be associated on occasion with insect attack or with adverse weather conditions. From this it follows that "blast" cannot be assigned to any one specific cause. It may be noted that the condition would not be expected to result from adverse factors, such as mineral deficiency or cereal root eelworm attack, which operate throughout the greater part of the life of the oat plant. This accords well with observation in the field.

I wish to thank Mr. H. E. Wells, Principal of the Staffordshire Farm Institute for providing facilities for this work, and Mr. W. E. Perry for the photographs.

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TRIAL OF ANTIBIOTIC PAINT FOR CONTROL OF BACTERIAL STEM CANKER OF PLUM

by A. E. W. BOYD *and* A. M. PATON*

Edinburgh and East of Scotland College of Agriculture

IN recent papers, Crosse (1957a and b) and Crosse and Bennett (1957) have outlined the present knowledge concerning the control of bacterial canker of cherry, and have described field trials in England with a streptomycin spray used as an alternative to Bordeaux mixture. According to these workers streptomycin was found to be effective against the leaf-spot phase of the disease and superior to Bordeaux mixture for this purpose. They found that an autumn spray of streptomycin reduced considerably the incidence of branch cankers in the following spring, but no conclusive evidence was obtained to recommend such a spray for the control of branch cankers which form the more destructive phase of the disease. In New Zealand Dye (1954) found that streptomycin, applied as a spray, gave a high degree of protection against "blast" of stone fruit in three field trials.

In Scotland, the well-known plum variety Victoria constitutes the bulk of the stone fruit trees grown. In older orchards, losses due to branch die-back are quite frequent, but much more serious are the losses in younger plantings due to cankers which almost invariably girdle the main stem below the crotch with the resultant death of the tree. Young trees up to six or seven years old appear to be particularly susceptible to this form of the disease and, as a result, growers now regard the replacement of losses as uneconomic. This can be illustrated by the following representative example. A grower planted 200 Victoria plum trees (on Myrobolan B stock) in an orchard in southern Scotland in 1951. The losses of trees from bacterial canker in each of the subsequent years were:

	1952	*1953*	*1954*	*1955*	*1956*	*1957*	*Total*
No. of dead trees ..	48	40	35	17	8	0	148

Thus, after a period of five years only about one quarter of the original planting remained alive. From these figures it appears that the disease shows its maximum effect during the first three years after the nursery stage.

The leaf phase of the disease must be considered the most important source of inoculum for the canker phase and, therefore, the most likely focus for an effective treatment. The potential inoculum, however, may be over any prolonged period of time. At monthly intervals over a period of three years attempts have been made to reduce the potential inoculum on the leaves and branches by means of streptomycin spray (220 i.u. per ml) in a trial involving 111 trees. It was hoped that an indication of the optimum period for a spray programme might be revealed, but in all cases neither leaf-spotting nor stem canker varied in incidence from that in the control trees.

The method of entry of the bacteria into the main stem is not clearly understood, but it is probable that wounds play a large part in allowing the organisms to penetrate. Where standard or half-standard trees are grown it is a common practice to stake the young trees for a number of years. In such cases wounds are frequently made by the "ties" or by the rubbing of the trees against the

* Now at Department of Bacteriology, University of Aberdeen.

stakes, and cankers have been frequently observed to originate at these points. In an attempt to protect the areas subject to abrasive action it was decided to apply paint to these parts. It was further considered that the effectiveness of such a measure would be enhanced by the incorporation of a suitable antibiotic in relatively stable form which would remain active for periods up to several months.

EXPERIMENTAL METHODS

A formulation of streptomycin hydrochloride as an emulsion paint was made available to us and was provided in the first instance with a streptomycin potency of 20,000 i.u. per ml. This was later reduced to 10,000 i.u. per ml. The paint was applied by brush to the bark where it dried quickly, forming a membrane which showed little sign of deterioration within six months of application. The activity of the underlying tissues was apparently unimpaired by the presence of the paint film. The stability of the antibiotic property was shown by the fact that, when painted on paper and exposed to weathering over winter, the material was still active against a test strain of *Pseudomonas* after approximately six months.

The site chosen for the experiment was in the walled garden of Dryden Estate belonging to the Horticultural Unit of the Edinburgh School of Agriculture. Victoria plum trees of considerable age grew inside the garden. These trees were all affected by bacterial canker to varying degrees and the site was thus considered suitable for the natural development of the disease. There is, as yet, however, no evidence to suggest any particular means of spread of the causal organism from tree to tree. One hundred two-year old half-standard Victoria trees on Myrobolan B stock, obtained directly from a commercial nursery, were planted within the garden. Paint was applied to twenty-five of these trees along 1 ft of the stem at the point of tying to the stake and the application was repeated every three months. Another twenty-five trees were staked only, while the remainder were untreated. The painting was carried out in January 1955 immediately after planting, and monthly examinations were made for signs of bacterial canker.

By July 1956, seven of the trees had died, but it was observed that these were distributed almost equally over the treatments, indicating that, if successful control were to be achieved, attention should be paid to the whole stem and not the "tie" area only. It was also noticed that neither of the fatal cankers on the painted trees originated at the painted area nor at any obvious point of abrasion. It was decided, therefore, to apply paint to the whole stem and crotch and include in the modified experiment treatments designed to show whether or not the causal organism could enter the normal bark, the painted bark, or both. With this in view, a proportion of the main stems were painted completely, and others incompletely, leaving an unpainted "collar" 6–8 inches long about two-thirds up the stem. The unpainted "collars" and their corresponding areas on the completely painted trees were covered with cotton wool saturated with a water suspension of a mixture of strains of the pathogen. The wool was prevented from drying out and weathering, by a covering of thin plastic sheeting. The cotton wool was re-inoculated at frequent intervals throughout the trial period. The strains of bacteria used were checked for pathogenicity at intervals during the experiment by inoculation of branches of trees of the same type and age as those in the trial.

EFFECTS OF TREATMENTS

The various treatments, and the incidence of fatal main stem cankers obtained over the first three years of the experiment, are shown in the Table. It seems clear that the number of fatalities involving painted trees, or areas of trees, is lower

than on unpainted trees. Of the six fatalities which involved painted areas, infection in five cases (two in Treatment 1, and three in Treatment 2) appeared to originate in the area of the union at about soil level. The appearance of these was atypical of bacterial canker and attempts to isolate a causal organism were unsuccessful. Whether or not weather conditions causing wind rocking were involved, since only trees which were unstaked at least during part of the experiment were affected, is not known. In the other case (Treatment 1) the tree died only three months after application of the paint, and it is highly likely that infection had occurred prior to painting. Another instance was observed where infection originated at a point on an unpainted branch immediately above the painted crotch, but the paint did not appear to inhibit the spread of the organism through the underlying tissue into the painted area. In this case the infected branch was removed before the main stem was invaded.

Effect of Streptomycin Paint Applied to Main Stems of Half-standard Victoria Plum Trees Planted in January 1955

Treatment	Original No. of trees	Trees dead by November 1957	Notes on death of trees
1. Unstaked. Unpainted until July 1956, then complete stem painted except at inoculated "collar".	26	7	1 dead before painting; 1 within 3 months of painting; 2 from collar inoculation; and 2 from cankers at union.
2. Staked. Painted under tie until July 1956. Then unstaked, and complete stem painted. Inoculated "collar" over paint.	25	5	2 dead before painting; 3 from cankers at union.
3. Unstaked. Unpainted.. ..	24	10	1 from canker at union.
4. Staked. Unpainted	25	15	3 from cankers at "tie".

In the attempt to infect trees by mass inoculation through the painted and unpainted bark, none of the painted "collars" (Treatment 2) became infected. In three out of twenty-five trees infection was obtained through unpainted bark (Treatment 1). In these cases the *Pseudomonas* strains used remained in contact with the bark, and were viable and unaffected by bacteriophage throughout the trial period of more than a year. Normal bark thus appears to be capable of resisting to a high degree the presence of a massive infection potential on its surface but, given optimum conditions as far as possible, the organism can infect the apparently unwounded main stem.

At least three of the fifteen typical cankers in unpainted and staked trees (Treatment 4) originated at the point where rubbing of the tie had occurred, suggesting that main stem infection may be encouraged by tying and staking. In other respects the number of deaths in the two unpainted treatments are very closely comparable.

From the data presented in the Table it may be concluded that the application of streptomycin paint to the main stem is associated with a reduction in main stem cankers.

It is not known at present whether bacterial-canker control is being achieved by the action of the streptomycin, or by the mechanical effect of the paint, or by both, but further extended experiments are in progress to investigate such factors. The preliminary results of these pilot experiments are reported now, since it is felt

that they may provide the basis of an easily applicable and relatively inexpensive method of control of bacterial canker which could be attempted immediately in any young plantation, in view of the extensive losses by this disease.

SENSITIVITY TESTS ON OPERATORS

During the conduct of these experiments particular attention was paid to the effect of the antibiotic materials on the health of the operators. For a period when the streptomycin spray and the emulsion paint were being extensively used, regular examinations by the Bacteriology Department of the Royal Infirmary of Edinburgh of the bacterial flora of the skin, nasal and oral cavities, and the intestinal tracts of the three persons most closely in contact with the antibiotic failed to reveal any resistant mutants. No form of allergy to the streptomycin was detected at any time during the trials.

SUMMARY

An emulsion paint containing streptomycin, when applied to the stems of young Victoria plum trees, reduced markedly the incidence of bacterial stem canker in a trial under field conditions in Scotland. The unwounded stem showed a high degree of resistance to penetration by high concentrations of the pathogen. The practice of tying young trees, leading to abrasions, may promote the infection of the stem. The health of the operators was unaffected by extensive use of the streptomycin preparations.

We wish to thank Glaxo Laboratories, Ltd. for the supply of the streptomycin formulations, and the Bacteriology Department of the Royal Infirmary of Edinburgh for the medical bacteriological examinations.

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SOIL-BORNE VIRUS DISEASES IN STRAWBERRY

by R. M. LISTER

Scottish Horticultural Research Institute, Invergowrie, by Dundee

THE possibility that some virus diseases may be transmitted through the soil has often been overlooked in investigating their modes of spread, but recently it has been shown that several diseases caused by soil-borne viruses are widespread and locally important in eastern Scotland and in England. Thus, raspberry ringspot virus, which causes raspberry leaf curl disease in some varieties of raspberry, has been found in the soil in many localities in the important raspberry growing areas of eastern Scotland (Cadman, 1956; Harrison, 1956); while raspberry yellow dwarf virus is known to occur in soils from several places in England (Harrison, 1958), and beet ringspot virus, which causes diseases in sugar beet, potatoes and some raspberry varieties, occurs in the soil in several places in eastern Scotland (Harrison, 1957).

Harrison found that strawberry is a host of raspberry yellow dwarf virus and beet ringspot virus, and further work has now shown that soil-borne viruses are widespread in cultivated strawberries, for they have been isolated from plants of several varieties from localities in western and eastern Scotland and in southern England. These viruses cause serious diseases in strawberry, which may have been noted before but assigned to other causes, for in some respects their symptoms resemble those caused by eelworms, severe crinkle virus, and the shallot aphid, *Myzus ascalonicus* Doncaster. They are thus probably even more widespread in strawberries than the available information indicates, and it is hoped that this account may assist in the identification of any further outbreaks, so that the importance of the problem can be better assessed.

All the viruses concerned infect a wide range of host plants, including common weeds: all persist for long periods in the soil, through which they are carried in some unexplained way, and none appears to be transmitted by an aerial vector. Unlike any other viruses reported to cause disease in strawberries, they can readily be transmitted mechanically from the leaves of infected strawberries to a number of herbaceous hosts, though apparently not to strawberry. The strawberry sap used in all transmission experiments was prepared by macerating pieces of leaf mixed with alumina and "Celite" in a small amount of water, afterwards diluting further before inoculation. Alumina, though not essential, appears to assist in preventing the inactivation of virus by the virus inhibitors present in expressed strawberry leaf sap (Thresh, 1956; Cadman, unpublished).

RASPBERRY RINGSPOT VIRUS

Raspberry ringspot virus was isolated from the strawberry varieties Talisman, Huxley and Cambridge Vigour. Affected plants of each variety were stunted and yields probably quite severely reduced. In all these varieties the virus caused yellow blotch symptoms of varying severity in the leaves: severely affected leaves were also crinkled (Plate I, 1).

Outbreaks of the disease were found in five localities in eastern Scotland and one in western Scotland and varied in size from discrete patches of a few plants to a thousand or more. Raspberry ringspot virus was easily transmitted from the leaves of affected plants by rubbing sap on to the leaves of *Chenopodium amaranticolor* Coste & Reyn., in which raspberry ringspot virus produces local lesions without the development of systemic infection.

Of forty-four Huxley plants with the yellow blotch symptom collected in various localities, the leaf sap of forty-one produced local lesions of the raspberry ringspot type in inoculated leaves of *C. amaranticolor*. The number of lesions produced varied between about five and one hundred per inoculated leaf. Altogether forty Huxley plants without the yellow blotch symptom were collected at the same time and from the same fields as the diseased ones, but no lesions resulted when leaf sap from them was rubbed on the leaves of *C. amaranticolor*. The sap from the leaves of plants of the varieties Talisman and Cambridge Vigour, with the yellow blotch symptom, also produced infections when rubbed on the leaves of *C. amaranticolor*.

The viruses thus isolated produced symptoms typical of those of raspberry ringspot virus in *Petunia hybrida* Vilm., *Nicotiana tabacum* L. var. White Burley, *Nicotiana rustica* L., *Phaseolus vulgaris* L. var. Prince, cucumber and *Datura stramonium* L. Their identity was established by plant protection tests in *Petunia hybrida* and by serological precipitation tests.

Aphid-borne viruses and raspberry ringspot virus sometimes occurred in the same plant. In one experiment eight Huxley plants with the yellow blotch symptom and twelve without were collected from the same field, and apterous strawberry aphids, *Capitophorus fragariae* Theob., were removed from each plant and transferred to runnerless *Fragaria vesca* L. seedlings. *F. vesca* plants infested with aphids from plants in each series later produced symptoms typical of Prentice's strawberry mottle and strawberry crinkle viruses (Prentice, 1952), but only sap from leaves of the eight Huxley plants with the yellow blotch symptom produced local lesions of the raspberry ringspot type on *C. amaranticolor*.

RASPBERRY YELLOW DWARF VIRUS

In the spring of 1957 plants of a stock of Cambridge Favourite strawberry, obtained from Devonshire, were planted at Mylnefield in a field experiment on virus spread. Graft tests on a small sample of the plants made in the glasshouse during the spring of 1957 indicated that they contained virus only of the strawberry crinkle type. By June, 1957 about 30 per cent of the plants in the field were very severely stunted (Plate I, 2) and their leaves were twisted, deformed and had yellow blotches. They produced few runner plants and their stolons were short, thick and brittle, and red in colour. These effects somewhat resembled those of the damage caused by the shallot aphid, *Myzus ascalonicus* (Dicker, 1950), or by eelworms (Anon, 1953), but unlike them they persisted and intensified during the summer, and some plants eventually died. Affected plants were distributed randomly in the plot, and the disease did not spread during the year.

To find out if these symptoms were associated with the presence of viruses, ten apterous strawberry aphids were removed from each of ten affected and ten normal-looking plants and allowed to feed on runnerless *F. vesca* plants in the glasshouse. Five out of the ten *F. vesca* plants in each series later produced similar symptoms of the strawberry crinkle type. However, sap from the leaves of all ten stunted plants produced a systemic virus infection when rubbed on the leaves of *C. amaranticolor* plants, but that from the ten normal-looking plants did not.

The virus concerned produced symptoms typical of those of raspberry yellow dwarf virus in White Burley tobacco, *Nicotinia rustica*, *Petunia hybrida*, French bean and *Datura stramonium*, and its identity was established by plant protection tests in *Petunia hybrida* and by serological precipitation tests using authentic material. All of thirty-six affected Cambridge Favourite plants, and none of thirty-five normal-looking ones, which were tested by rubbing sap on to the leaves of *C. amaranticolor*, proved to be infected with the raspberry yellow dwarf virus.

It was later found that about 30 per cent of plants from the same stock of Cambridge Favourite planted in a locality in Angus in the spring of 1957 were similarly affected, and raspberry yellow dwarf virus was also isolated from some of these. From the distribution of the affected plants, both in this planting and the one at Mylnefield, it appeared that they had been infected, though symptomless, when planted.

While these investigations were in progress, Harrison (1958) isolated raspberry yellow dwarf virus from strawberry plants of the varieties Cambridge Favourite and Deutsch Evern, which had been collected in Kent by Dr. A. F. Posnette of East Malling from near an outbreak of yellow dwarf disease in raspberries. These plants had similar symptoms to those described above.

In November, 1957, a few strawberry plantings in Lanarkshire were examined for possible outbreaks of soil-borne virus diseases, at the suggestion of Mr. R. D. Reid of Auchincruive, who had noted persistent stunting in plants in some of them during 1957. At two farms on rather heavy soils, patches of severely stunted plants of the variety Talisman were seen in plantings which were otherwise excellent. At one of the farms well over a thousand plants were involved, but at the other only a very few plants appeared to be affected. Examination of the roots of affected plants did not reveal the symptoms of attack by red core, *Phytophthora fragariae* Hickman. At the time of this investigation the leaves of the stunted plants were normal, although rather small, but all those sampled proved to be infected with raspberry yellow dwarf virus, which was identified by serological precipitation tests after transmission to *Petunia hybrida* plants.

The distribution of the infected plants seen in Lanarkshire strongly suggests that raspberry yellow dwarf virus is endemic there, though it has not previously been found to occur in soils in Scotland. This is perhaps not surprising since the virus seems to be associated with heavier soils than either raspberry ringspot virus or beet ringspot virus, and previous investigations have been largely restricted to the eastern counties where much of the fruit is grown on light loams. It may be that raspberry yellow dwarf virus has been introduced into soils in eastern Scotland in the past, but it has failed to become established. The factors influencing their establishment in the soil, and the way in which they are transmitted, are among the most important problems awaiting solution in the study of soil-borne viruses.

I wish to acknowledge the assistance of Mr. D. H. Turner, of the Edinburgh and East of Scotland College of Agriculture, in field investigations. I am indebted to Miss Patricia Duncan for technical assistance, and to Mr. J. Sunderland for the photographs.

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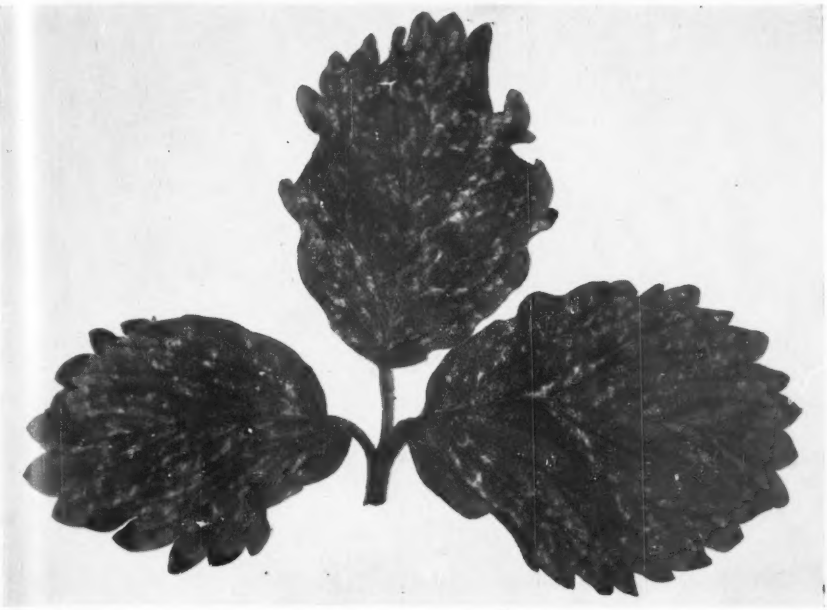


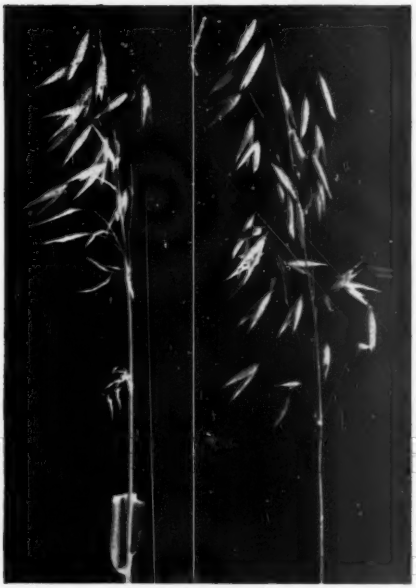
1. Leaf of Huxley strawberry infected with raspberry ringspot virus.

Photos: Scottish Horticultural Research Institute

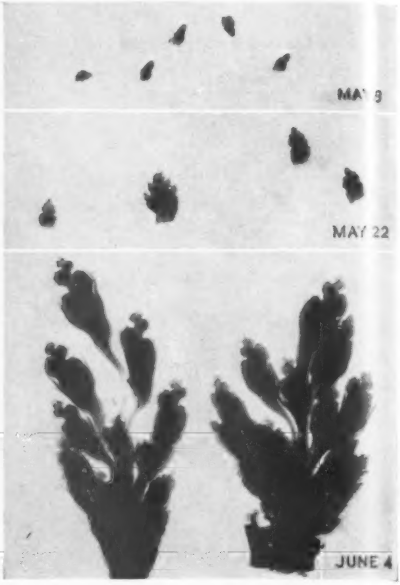
2. Maiden plants of Cambridge Favourite strawberry; (*left*) healthy, (*right*) infected with raspberry yellow dwarf virus.

PLATE I

"BLAST" OF OATS (*see pp.* 85-7)



1. Panicle from plant defoliated on June 19, and from control (*right*). Both photographed on July 9.



2. State of primordia at 2-leaf stage, May 8; at 4-leaf stage, May 22, and at 5-6 leaf stage, June 4. All ×8.

PLEOSPORA BETÆ ON WILD BEET (*see p.* 113-4)

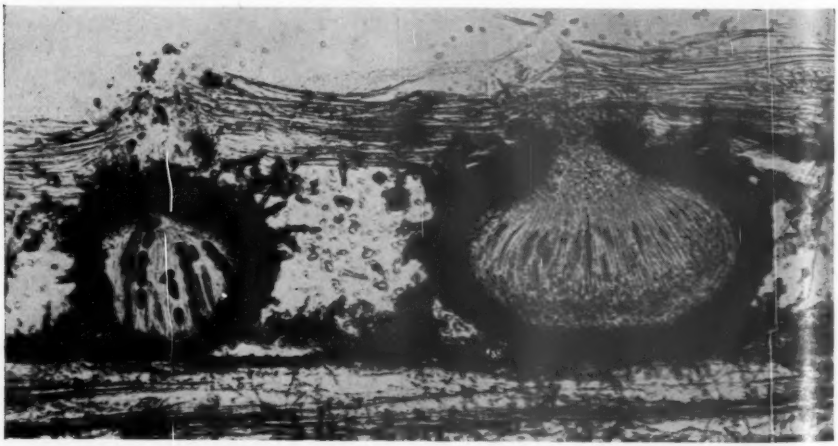
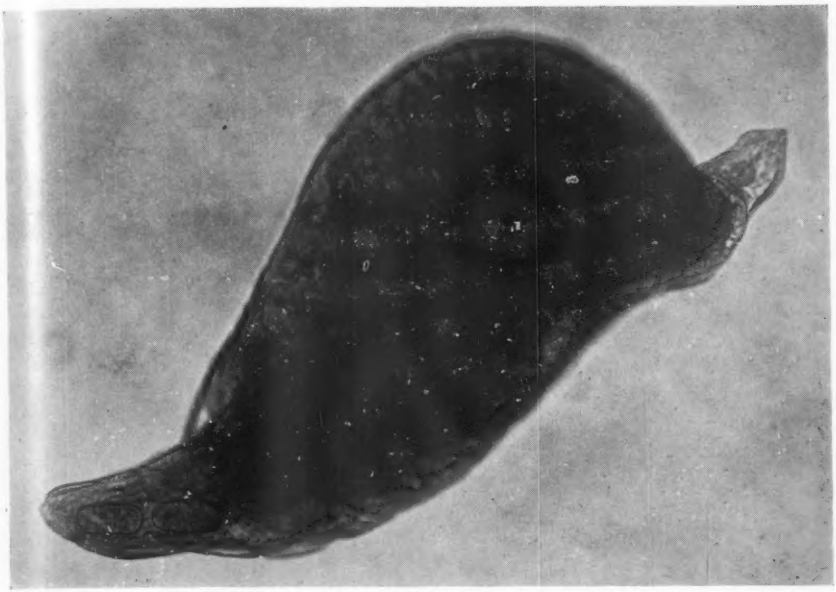


Photo: Rothamsted Experimental Station

3. Section through two perithecia of *Pleospora betae*. ×200.

PLATE II

A NEMATODE GENUS NEW TO EUROPE (*see p.* 114)



1. Female of *Nacobbus* sp. ×125.

Photos: Rothamsted Experimental Station

2 Tomato roots with galls containing *Nacobbus* sp., showing characteristic proliferation of lateral roots. Actual size.

PLATE III

1. Systemic brown rings on *Anemone coronaria.*

2. Whitish local necrotic dots on *Gomphrena globosa.*

3. Diffuse local chlorotic spots on *Chenopodium amaranticolor.*

4. Large black local necrotic rings on *Aquilegia caerulea.*

PLATE IV

ANEMONE BROWN RING—A VIRUS DISEASE

by M. HOLLINGS

Plant Pathology Laboratory, Harpenden, Herts

DURING a visit in November 1956 to anemone fields in the Cheddar area, which I made with Mr. L. Ogilvie and Mr. B. B. Till (N.A.A.S. Plant Pathologists at Bristol), one anemone plant was found with symptoms differing from any previously seen. This plant was brought back to the Plant Pathology Laboratory for investigation.

The symptoms consisted of a number of brown rings, 2–4 mm diameter, with pale tan centres, on the older leaves. During the next month these rings darkened considerably, some becoming black, while many enlarged and coalesced to form large dark brown areas enclosing a few pale spots, and leading to the total necrosis of these older leaves. The younger leaves showed only a slight general chlorosis; the middle leaves bore faint chlorotic spots which slowly developed brown margins, and increased in severity as growth proceeded. The plant steadily declined in vigour and by mid-February was moribund: no flowers were produced. *Botrytis* was the only organism isolated from the plant.

As the disease seemed likely to be due to virus infection, mechanical inoculations from leaves showing conspicuous rings were made to anemone seedlings, previously checked by inoculation to *Chenopodium amaranticolor* Coste & Reyn. and believed to be virus-free. "Celite" abrasive and M/15 phosphate buffer (pH 7·0) were used, and test plants were kept in darkness for 1–2 days before inoculation. After 7–8 weeks faint brownish rings appeared on the older non-inoculated leaves of two out of fourteen inoculated anemones; symptoms on these plants subsequently developed in a similar manner to the original infected anemone.

Plate IV, 1, shows the systemic brown rings on older leaves ten weeks after experimental infection.

In subsequent inoculations seven out of thirty-eight anemones were infected, though only after repeated inoculations to the same plants; host-range and other tests were made from sap from the older leaves of these plants. The virus concentration in the younger, almost symptomless, leaves of infected anemones was very much lower than that of the older leaves or the roots.

EXPERIMENTAL HOST RANGE

Chenopodium amaranticolor proved a useful indicator plant for anemone brown ring, showing local diffuse chlorotic spots, 2–2½ mm diameter, after 8–12 days; these lesions later enlarged slightly, and developed reddish-brown necrotic margins (Plate IV, 3).

All other test plants were checked for infection by inoculation to *C. amaranticolor* after 3–4 weeks, and again after about 2 months.

The observations were as follows:

Amaranthus caudatus L. Few, faint brown local rings after 10 days.

Gomphrena globosa L. Few, whitish local necrotic dots after 12–15 days (Plate IV, 2).

Tetragonia expansa Murr. Few, faint diffuse local chlorotic spots up to 5 mm diameter after 2 weeks.

Portulaca oleracea L. Carried without symptoms, in low concentration, in the inoculated leaves.

Aquilegia caerulea James. Prominent local blackish necrotic rings, up to 5 mm diameter, after 10–14 days, later enlarging slightly or developing concentric margins. Each lesion had a central necrotic dot. Virus present in low concentration (Plate IV, 4).

Lathyrus odoratus L. Carried without symptoms in most inoculated leaves; occasional leaves developed tiny local necrotic dots after 2½ weeks.

Lycopersicon esculentum Mill. (var. Kondine Red). Few, faint local chlorotic spots after 12 days; virus present in very low concentration.

No symptoms could be seen on any of the following plants, nor could the virus be recovered from either inoculated or un-inoculated leaves:

Ageratum conyziodes L.
Alonsoa warscewiczii Regel.
Antirrhinum majus L.
Apium graveolens L.
Beta vulgaris L.
Brassica pekinensis Rupr.
B. rapa L.
Callistephus chinensis Nees.
Celosia argentea L.
Chrysanthemum morifolium Ramat.
Convolvulus tricolor L.
Cucumis sativus L.
Datura stramonium L.
Dianthus barbatus L.
Fagopyrum esculentum Moench.
Fumaria officinalis L.
Helianthus annuus L.
Hyoscyamus niger L.
Lactuca sativa L.
Lythrum salicaria L.
Nicotiana affinis Moore.
N. clevelandii Gray.
N. glutinosa L.
N. rustica L.
N. tabacum L.
Papaver somniferum L.
Passiflora caerulea L.
Petunia hybrida Vilm.
Phaseolus vulgaris L.
P. coccineus L.
Phlox drummondii Hook.
Physalis floridana Rydb.
Phytolacca americana L.
Plantago lanceolata L.
Primula malacoides Franch.
Reseda odorata L.
Senecio vulgaris L.
Spinacia oleracea L.
Stellaria media (L) Vill.
Trifolium repens L.
Tropaeolum majus L.
Verbena hybrida Voss.
Vicia faba L.
Vigna sinensis Savi.
Zinnia elegans Jacq.

Anemone coronaria was not easily infected by mechanical inoculation, and infected plants usually died within a few weeks after the appearance of symptoms —especially in winter. No other host plant was found in which the virus reached a satisfactory concentration, and it proved difficult to maintain the virus. In fact, in the summer of 1957 a number of inoculations to anemone failed, and the virus was unfortunately lost.

PROPERTIES IN VITRO

The properties *in vitro* were assayed in a limited number of tests from anemone sap to *C. amaranticolor*.

Dilution end-point: the maximum infective dilution of infected anemone leaf sap was 1/100, using mature leaves with well-developed symptoms; the number of local lesions on twenty-four leaves of *C. amaranticolor* at various dilutions with phosphate buffer were:

Dilution	1/2	1/5	1/10	1/25	1/50	1/100	1/250	1/500
No. of lesions ..	83	18	11	4	2	1	0	0

Sap from young infected leaves withstood dilution only to 1/5 or 1/10.

Thermal inactivation point: expressed anemone sap, diluted 1/2 with buffer, was infective after 10 minutes' heating at 60°C, but not at 65°C; the numbers of local lesions per 24 *Chenopodium* leaves at 40, 50, 55, 60, 65 and 70°C respectively were 27, 35, 15, 3, 0 and 0, compared with 67 lesions from the unheated control inoculum.

Longevity in vitro: expressed anemone sap, diluted 1/2 with buffer, was still infective after 10, but not 15, days at room temperature (about 18°C), though over 96 per cent of the infectivity was lost in the first 24 hours.

The virus was inactivated within 15 minutes by 50 per cent ethyl alcohol, or by 10 per cent "Teepol" detergent; and within 2 minutes by 5 per cent Na_3PO_4 solution.

The virus is clearly not very stable in anemone sap.

APHID TRANSMISSION TESTS

Adult apterous *Myzus persicae* (Sulz), *Aulacorthum solani* (Kltb.) and *Macrosiphum euphorbiae* (Thos.) failed to transmit the virus from anemone to anemone; 20–30 aphids per plant were used, the acquisition feeds were 2–5 minutes and test feeds 1–2½ hours in one series of tests, and both feeds were 24–48 hours in another test.

SOIL TRANSMISSION TEST

Two infected anemone plants were grown in large trays of John Innes potting compost No. 2, surrounded by twelve healthy anemone seedlings. No symptoms of the disease could be seen in the test plants over the following ten months, though both the infector plants died during this period. This failure to spread under test conditions in no way excludes soil transmission, since spread of some soil-borne viruses appears to occur only with certain naturally-infected soils (Harrison, 1957).

SEROLOGICAL TESTS

The concentration of ABR virus in every plant so far tested offered little prospect of obtaining an antiserum, especially as little material was available. Precipitin tests were, however, made with infected anemone sap against the following plant virus antisera: the dilution at which each antiserum was used is given in brackets. Anemone mosaic (1/8), bean yellow mosaic (1/20), barley false stripe (1/16), broad bean mottle (1/20), carnation ringspot (1/10), hyoscyamus virus III (1/10), hydrangea ringspot (1/20), lucerne mosaic (1/10), pelargonium leaf curl (1/10), potato virus X (1/20), potato virus Y (1/10), tobacco mosaic (1/20), tobacco necrosis (1/20), tomato black ring (1/10), and normal serum control. Controls were also run with healthy anemone sap and test antisera. All saps were heat-clarified for 10 minutes at 35–40°C, then chilled and centrifuged at 7500G for 20 minutes, and serial dilutions of 1/2 to 1/64 prepared with saline. One ml of antiserum was mixed with 1 ml of diluted sap, and tubes incubated at 37°C for 10 hours. No specific precipitates occurred with any of the sera tested, though these all reacted with their homologous antigens.

DISCUSSION

The experimental evidence confirms that the disease is due to a virus which appears to differ from any so far described; the name anemone brown ring is proposed for the virus and the disease caused.

There is at present no clear evidence of the relationship of ABR to any particular group of viruses, but certain features suggest similarities to the tobacco rattle (potato stem-mottle) type of virus; symptoms of ABR on aquilegia closely

resemble those illustrated for tobacco rattle on this host (Schmelzer, 1957), but tobacco mosaic also produces this type of symptom on aquilegia. The properties *in vitro* are more like those of the soil-borne ringspot viruses, but the host range of ABR is very much more restricted than that of tobacco rattle or the soil-borne ringspot viruses.

Only one infected plant has so far been noticed in the anemone fields of Cheddar, and none in Devon or Cornwall, so that the virus would seem to be uncommon. So far, no infected anemones have flowered, though whether this is in itself a symptom or merely a consequence of reduced vigour is uncertain; but the steady decline of infected plants could lead to considerable losses if the disease became prevalent.

I should like to thank Dr. Kassanis of Rothamsted for carrying out the serological test with his antiserum to barley false stripe.

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ALDRIN AND *GAMMA*-BHC FOR THE CONTROL OF WIREWORM DAMAGE TO POTATOES

by F. E. MASKELL

National Agricultural Advisory Service, Cambridge

BEVAN and Bryden (1956), reporting experiments using aldrin to control wireworms in potatoes, stated that 2 lb of aldrin per acre applied to the soil before planting gave a reasonable reduction of damage at population levels below ½ million wireworms per acre. They quoted the results of work (for which I was responsible) in the Eastern Counties. In this experiment, with an initial population of 950,000 wireworms per acre, 3.36 lb per acre of aldrin reduced the percentage of damaged tubers only from 74 to 37. It was this disappointing result with a high wireworm population which led to the idea that aldrin might not be as efficient as *gamma*-BHC and suggested the need for a direct comparison between the two insecticides. Although *gamma*-BHC could not be used on a commercial potato crop owing to risk of taint, it was thought that potatoes would give a more sensitive indication of treatment effects than estimates of wireworm populations based on standard sampling methods with their high error (Anon. 1944).

The ploughing up of a heavily infested piece of old grassland for experimental plots at Anstey Hall offered scope for such a trial on potatoes, and gave the opportunity of comparing dust and high volume spray applications, on which Bevan and Bryden had only limited data.

The soil was an organic sandy loam and the grass was ploughed in April 1955. The wireworm count immediately before ploughing was 1,475,000 per acre, or 1,075,000 exceeding 4·9 mm in length (the size group on which advice is normally

based). The wireworms were extracted from the soil by the flotation process described by Cockbill, Henderson, Ross and Stapley (1945). The plots were each 16 yd × 5 yd arranged as 4 × 5 randomized blocks with the following treatments:

1. Control—no treatment.
2. Aldrin dust (1¼ per cent) at 1¼ cwt per acre, giving 2·1 lb aldrin per acre.
3. Aldrin miscible liquid (30 per cent) at 6 pints per acre, giving 2·25 lb aldrin per acre.
4. *Gamma*-BHC Wireworm Dust (0·45 per cent) at 1¼ cwt per acre, giving 0·76 lb *gamma*-BHC per acre.
5. *Gamma*-BHC miscible liquid (10 per cent) at 6 pints per acre, giving 0·75 lb *gamma*-BHC per acre.

The dusts were broadcast by hand, and the sprays applied by knapsack sprayer at approximately 100 gallons per acre on April 20, 1955 after the ridges had been split for planting. Care was taken to apply the materials as evenly as possible.

A compound potato fertilizer was broadcast at 10 cwt per acre before planting Majestic seed. In order to obtain additional information on residual effects and taint, potatoes were planted on the same site in the following year without the application of any insecticide or fertilizer. The potatoes were planted on April 21 in 1955 and March 26 in 1956 and were lifted on October 12 in 1955 and October 20 in 1956.

Each plot consisted of six rows, and the two outside rows and a length of 1 yd at each end of each plot were discarded. Ten plants were lifted at random from the central area of each plot for detailed examination and these plants were used to obtain the following records: the number of tubers; the weights of ware (those not passing through a 2-inch riddle) and chats; the number of tubers damaged by wireworms, slugs and cutworms; the total number of wireworm, slug and cutworm holes before peeling, and the number of wireworm holes visible after peeling. In addition the total yield, including the ten harvested roots, was recorded for each plot.

In 1955 the total number of tubers examined per treatment ranged from 587 to 684, and in 1956 from 390 to 530—including the very small ones.

The results are given in the Table.

Treatment	Year	Percentage of Tubers Damaged		Total plot Yield (lb)
		by Wireworms	*by Slugs and Cutworms*	
Control ..	1955	76·2	10·4	751
	1956	65·4	30·6	1225
Aldrin Dust ..	1955	30·2	3·7	780
	1956	28·2	25·8	1258
Aldrin Spray ..	1955	29·8	1·4	782
	1956	30·0	15·6	1294
Gamma-BHC Dust	1955	23·7	1·3	760
	1956	23·8	17·9	1316
Gamma-BHC Spray	1955	24·1	1·7	734
	1956	33·2	29·5	1243

In 1955, because of the very dry weather, the yields were low and the proprotion of ware potatoes small; a heavy infection of scab made the examination for

wireworm holes before peeling difficult. In contrast 1956 was very wet, particularly in the latter months, and consequently the ware weight and the proportion of tubers damaged by slugs showed a marked increase. Because of the difficulty of distinguishing between slug and cutworm damage, these are recorded together in the Table. The percentages of tubers damaged by wireworms and slugs and cutworms were analysed after angular transformation of the figures, and in both years the aldrin and *gamma*-BHC treatments gave a significant reduction of wireworm damage, though 20–30 per cent of the tubers were holed even on the treated plots. There were no significant differences between aldrin and *gamma*-BHC or between dusts and sprays. There were no significant differences from the control in the number of tubers, the total weight and the ten roots ware weight.

Although the percentage of tubers damaged is the simplest and most satisfactory measure for practical purposes, the differences in the number of holes per tuber is more striking. In 1955 this averaged 5·58 on the untreated, and ranged from 0·55 to 0·98 on the treated; while in 1956, the corresponding figures were 3·51 and 0·69 to 1·04. The reduction of tubers damaged by wireworm after peeling was 8 per cent in 1955 and 7 per cent in 1956, showing that most of the holes were deep and no useful information was obtained by this additional work. As in the previous trial, quoted by Bevan and Bryden, both treatments significantly reduced slug and cutworm damage in 1955. It was reasonable to expect a reduction in this year when much of the damage was due to cutworms but in 1956, when slugs were mainly responsible, the reduction was much less and not significant.

The comparable effect of *gamma*-BHC and aldrin confirms the work of Merrill (1952). The relatively poor results of either insecticide against high wireworm populations may possibly be due to inadequate mixing of the insecticide with the soil.

In October 1955, the plots were resampled individually for wireworms (ten 4-inch cores per plot) after the potatoes had been lifted. The numbers on the control plots had dropped to 225,000 per acre, and those on the treated plots to about 50,000 per acre as an average for all treatments. In January 1957, the population on the controls was 25,000 per acre and for treated plots 9,400 per acre. It is surprising that, with such low populations, damage to the extent of between 24 and 33 per cent was found on the treated plots in both years.

Tasting tests were carried out in both years amongst colleagues using the "triangular" test, and samples were also sent to a trained tasting panel who used a scoring system. The results obtained by both panels were in agreement; in the 1955 tests, the BHC-treated samples had significant degrees of taint, whereas in 1956 (18 months after applying the treatments) the residual effects of the BHC did not differ significantly from those of the controls. No off-flavour of the samples from the aldrin dust and spray treatments was recorded in either year.

This work was carried out under the general supervision of Dr. H. C. Gough, and I am greatly indebted to him for every help and encouragement with the preparation of this paper.

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DIELDRIN SPRAYS AS A CONTROL FOR FRIT FLY IN SPRING OATS

by J. H. FIDLER

National Agricultural Advisory Service, Cardiff

IN 1953, the entomologists of the National Agricultural Advisory Service started experiments using four sprays of 25 per cent miscible DDT at 4/5 gallon per 100 gallons of water per acre to control damage caused to spring oats by frit fly (*Oscinella frit* L.) larvae. In 1954, near Usk, Monmouthshire, small plots were sprayed twice in the tiller stage and twice after panicle burst, using either DDT, dieldrin or parathion. The plots were sown at three different dates, April 6 and 20, and May 4. In spite of the late sowing dates attack was very light, and only 15 per cent of the shoots were damaged in the untreated plots sown on May 4. Both DDT and dieldrin gave good control of tiller attack, reducing it to less than 1 per cent; parathion gave little control. Only DDT gave an appreciable reduction of second generation grain damage.

Since the later applications can hardly be considered practical for the farmer, as considerable damage would be caused by the spraying machine, this line of investigation was discontinued and attention focused on the control of the first generation. The assumption was made that if fewer flies emerged in a field from the first generation, there might be less to lay eggs in developing panicles.

Some early failures in obtaining a good control of the first generation with dieldrin seemed to arise from lack of knowledge of the best time to apply this spray in relation to adult frit fly emergence and plant size at the time of spraying. Trials were therefore laid down in 1955 and 1956 by applying a single spray on four or five different dates, each separated by an interval of a week. In 1957, this was repeated using two dates of sowing separated by twenty days. In lay-out the trials consisted of plots about 10 yards long by one drill width (thirteen rows). There were three replicates of each spray treatment, the position of the plots being randomized within each block.

A spray of ½ gallon of 15 per cent miscible dieldrin plus 8 oz manoxol O/T. per 100 gallons water per acre was applied by knapsack. The variety Star was sown in 1955, Sun II in 1956 and S.84 in 1957.

Damage by frit larvae was estimated in 1955 by taking fifty plants at random from each plot and dissecting them in the laboratory. In 1956, when the attack was exceptionally heavy, excessive time was taken in the examination of so many plants. It was then found that a sample of fifty plants was giving a within-plot standard error of 11 per cent, while reducing sample size to twenty-five plants would give 15 per cent error. Thereafter, the smaller sample was adopted. The untreated control plots were examined each week, and experience has shown that maximum damage estimated in this way is reached when the plant has just developed its fifth leaf on the main stem. When this stage was reached similar samples were collected from all sprayed plots and examined for damage.

EFFECTS OF SPRAYS ON FIRST GENERATION ATTACK

The results of these counts are plotted in Fig 1; the size (leaf number) and percentage of shoots attacked in the control plots are shown as continuous curves. The percentages of damage in the sprayed plots were all estimated in June at the

dates indicated by arrows, but are shown as columns placed over the dates when the plots were sprayed. In most cases the first spray, applied when the crop had been sown only two to three weeks, was generally less effective than that applied a week later, this latter treatment being the most effective of all. The exception was in 1956, when the attack was very heavy and started when the plants had hardly emerged from the soil. Tiller production was extensive and, as shown in Fig. I, does not give a true indication of the effects of these two applications of insecticide. Thus in the first spray 5·7 shoots per plant were attacked out of a total of 7·7, and in the second 6·4 out of 8·0. In the unsprayed plots 8·6 were

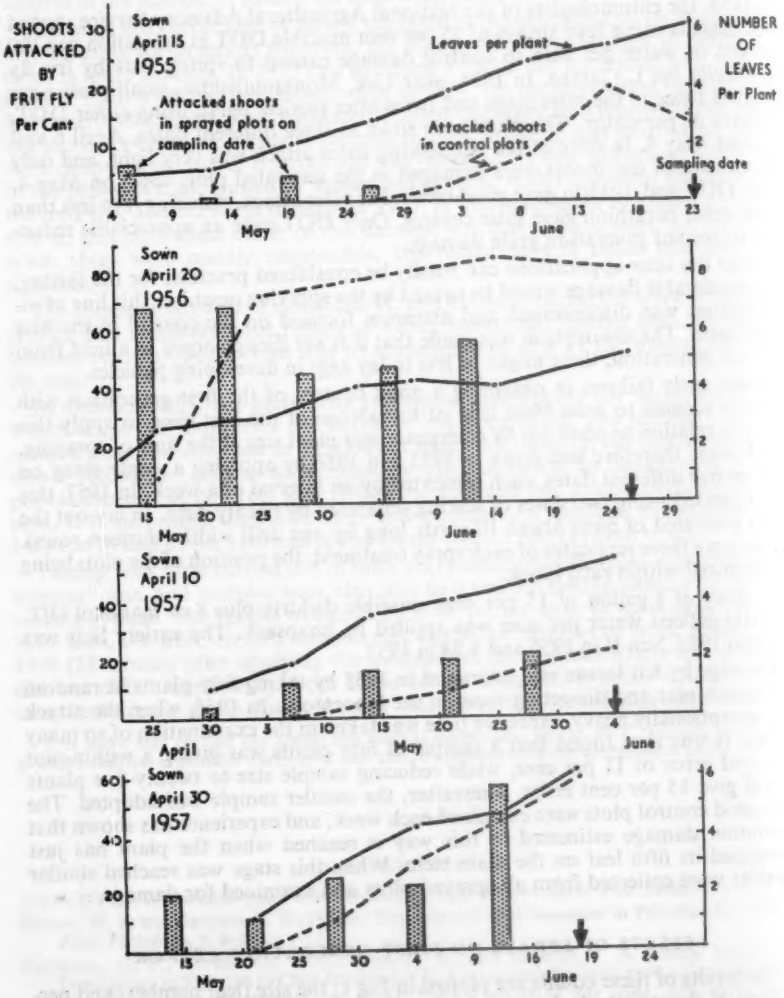

Fig. 1. Development of first generation frit fly attack on untreated plots and on plots sprayed once with dieldrin, 1955–57.

attacked out of 9·9. Spray applications had, therefore, considerably checked further attack and the need for continued production of new shoots which were themselves subsequently attacked.

In 1955, all four sprays were applied before the attack started, while in 1956 all were applied during or after the main attack. Such also was the case in the second sowing in 1957. In the later sprays, in these two trials, it was noticeable that the ultimate number of attacked shoots found in sampling at the final date was less than had been found in the unsprayed plots at the date when the sprays were applied. Since there is no reason to believe that the sprayed and unsprayed plots differed by this amount before treatment it is concluded that some shoots originally classed as damaged were ultimately classed as healthy. Since examination was carried out in the laboratory by dissection, even very early damage could be detected. If the larva causing this damage was killed by insecticide, the shoot could recover and the early damage be later unrecorded. This supports the observation by Walker (1953) that schradan and parathion can kill the larva inside the shoot; a similar suggestion was made as a result of work on seed dressings (Fidler and Webley, 1956). In the earlier sown crop in 1957 the later sprays were less effective than the first, and it must be concluded therefore that the shoots in these plants had reached a stage when they could no longer recover by the time the attack developed.

YIELDS FROM SMALL PLOT TRIALS

In 1956 and 1957, yields obtained from these plots were estimated by hand-cutting an area 6 yards long by 8 drills wide from the centre of each plot. These were threshed and cleaned and the moisture corrected to 15 per cent. The results as cwt per acre are given in Table 1.

TABLE 1

Yields from Small Plot Trials in cwt per acre at 15 per cent Moisture

Spray treatment	1956	1957 *normal sowing*	1957 *late sown*
Untreated	3·7	6·3	0·9
First spray	5·0	7·1	3·2
Second spray	4·2	4·8	1·8
Third spray	9·6	5·0	1·3
Fourth spray	7·2	6·6	1·0
Fifth spray	5·6	5·3	1·1

All yields were disappointingly low; but in 1956 and the late sown 1957 crop, when the attacks were extremely heavy, the yields were increased by the most effective sprays to about three times that of the untreated plots. In the earlier crop in 1957 the first generation attack was small in comparison with the attack on the grains, and its control therefore would have less effect on yield. It must be remembered that these crops were all sown rather late in the season and also suffered a very heavy second generation attack. In 1956, this was extremely heavy, 69 per cent of the grains being damaged in the untreated plots, while in 1957 the figures were 41 and 47 per cent respectively. In small plots, as used in these trials, those treated with an insecticide are surrounded by a much larger area of untreated oats from which frit flies, no doubt, migrate into the treated plots. Some evidence of the degree of migration into much larger plots is available from the large plot trials described below. In small plots grain attack

in the treated and untreated plots did not differ significantly. In the late sown crop in 1957 the final spray was applied on June 4 and the crop harvested on August 16. The grain attack on these plots was 39·7 per cent compared with 47·5 per cent on the other plots; a difference which is not significant, but it may show a tendency for the dieldrin to last sufficiently long enough, when applied at a date at which not too much damage is done to the crop by the machinery, to reduce to some extent the second generation attack.

TRIALS ON A FIELD SCALE

As a comparison with these small plot trials a parallel series of field applications was undertaken. In these the dieldrin spray was applied at $\frac{1}{8}$ gallon in about 12 gallons of water per acre by a tractor sprayer. The fields were divided into plots $\frac{1}{2}$–$1\frac{1}{2}$ acres in extent, part only being sprayed. In 1954 and 1955, a single spray of DDT at $\frac{4}{5}$ gallon per acre was applied as a comparison with dieldrin, while in 1957 only dieldrin was used. The results are given in Table 2.

TABLE 2

Results of Field Trials, 1954, 1955 and 1957

Site	Fritted Shoots per cent			Fritted Grains per cent		
	Dieldrin	*DDT*	*Control*	*Dieldrin*	*DDT*	*Control*
Monmouth Sown 13.4.54 Sprayed 10.5.54	0·6	2·7	3·3	4·4	3·2	2·8
Mitchel Troy Sown 14.4.55 Sprayed 9.5.55	7·3	12·4	27·6	10·8	10·3	11·5
Rhos (a) Sown 11.4.57 Sprayed 29.5.57	26·5	—	45·7	10·0	—	20·0
Rhos (b) Sown 11.4.57 Sprayed 29.5.57	21·4	—	33·9	11·0	—	18·0
Bedlinog Sown 17.4.57 Sprayed 29.5.57	25·8	—	45·6	—	—	—

In neither 1954 nor 1955 did DDT give such a good control of tiller attack as dieldrin; and although DDT is a rather cheaper material, it was not considered worth continuing with its use as a single spray. No direct comparison is available of a single dieldrin spray against two of DDT as described by Empson (1958); but an indirect comparison was made in 1957 when, immediately adjoining the small plot trial described above, another plot was sown with the variety S.221 on the same dates, but sprayed twice in the earlier stages with DDT. This variety is rather less susceptible to tiller attack than S.84, and the untreated plots showed 12 and 32 per cent of attacked shoots on the early and late crops respectively. In the DDT sprayed plots the figures were 8 and 13 per cent respectively, which is only about half the percentage reduction in attack obtained with the best single dieldrin sprays.

In the 1954 and 1955 field trials the sprays were applied three to four weeks after sowing, and this reduced the attack to about one-fourth of that on the untreated plots. In 1957, the sprays were applied six to seven weeks after sowing,

when the attack was well developed. Tiller attack was, on average, reduced by only 41 per cent compared with untreated plots, but was still useful considering the size of the attack.

In all except the Bedlinog trial, second generation attack was estimated by taking 100 heads at random from around the centre of the plots, and examining samples of 100 main and 100 bosom grains for damage. In neither the 1954 nor 1955 trials was there any significant difference in grain attack on the plots where different treatments had been applied in the early stages. In both these trials the plots were, however, rather long and only about 15 to 25 yards wide. It seems, therefore, that this distance is not sufficiently large to prevent the flies from migrating from the adjoining unsprayed plots. In 1957, the two fields at Rhos were about 3½ acres, and the half-field plots practically square. In these it seemed that frit fly migration was greatly reduced, second generation attack being in about the same ratio as those of the first.

It was possible to estimate yields only at Mitchel Troy in 1955. This was done by cutting a single swath down the centre of each plot with a combine harvester and weighing the grain. One of the dieldrin plots had been damaged by cattle and was therefore discarded. The dieldrin plots gave 16·7, the DDT 16·4, and the controls 10·6, cwt of grain per acre. Since the second generation attack on this trial had not been controlled, the increased yield in both DDT and dieldrin sprayed plots was most encouraging.

CONCLUSIONS

Four years of field and small plot trials enable some conclusions to be drawn on the use of dieldrin sprays against frit attack on late sown spring oats; the insecticide can be used as an efficient preventative if applied about three to four weeks after sowing, this being the most effective time of application for crops sown early in April.

With later sown crops it is possible to use a dieldrin spray even after some damage has become apparent, since there is still time for the oat plant to recover from some of the effects of the attack. An application at any time up to the 3½ leaf stage, or the first week in June, will give a reasonably good return.

There is good evidence that a single spray of dieldrin gives a better control than a single one of DDT, but it is doubtful if it is any better than two such sprays of the latter insecticide; however, two DDT sprays cost 2½ times as much as a single dieldrin treatment.

Evidence is incomplete as to the effectiveness of such sprays, applied early in the development of the plant, in reducing second generation attack on the grain. There is, however, some suggestion that if the field is large enough, migration from surrounding oats crops or grass fields may be limited, but more observation of frit fly migration is needed.

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WIREWORMS AND BASIC SLAG

by ALAN IBBOTSON

School of Agriculture, King's College, Newcastle-upon-Tyne

IN 1956 the "classical" Tree Field grazing experiment at Cockle Park in Northumberland was ploughed up. The field had been divided into ten fenced, 250 yd × 50 yd plots, side by side, each of which had received more or less the same treatment since 1896 and had developed its own soil type and sward. The original object of the experiment was to compare the live-weight gain of sheep grazed on the plots which were treated as follows:

1. 300 lb high protein concentrate fed to sheep annually until 1950, no other treatment.
2. 3½ cwt mineral phosphate every 3 years.
3. 10 cwt basic slag every 6 years.
4. 5 cwt basic slag every 3 years.
5. 7½ cwt superphosphate every 3 years.
6. No treatment.
7. Slag as Plot 4 and 100 lb muriate of potash every 3 years.
8. Slag as Plot 4 and 1 ton common lime every 3 years.
9. Slag as Plot 4 and 112 lb nitrate of soda every 3 years.
10. 609 lb dissolved bones every 3 years. (This plot fell into disuse and was not considered in the present observations.)

The wireworm populations of the plots were sampled in November 1955 just before the field was ploughed. Thirty 2½-inch cores were taken from each plot, in two random series of fifteen each, respectively from the ridges and furrows which crossed the field, and washed individually through an apparatus similar to that used by Salt and Hollick (1944). The approximate population per acre, by plots,

TABLE 1

Size, Distribution and Probable Limits of Population of Wireworms per Acre from Tree Field Plots

Plot No.	Ridges				Furrows			
	No. of Wireworms in Sample			*Thousands per acre*	No. of Wireworms in Sample			*Thousands per acre*
	0–5 mm	*6–10 mm*	*over 10 mm*		*0–5 mm*	*6–10 mm*	*over 10 mm*	
1	0	0	2	100–220	0	0	2	100–220
2	1	1	1	150–300	0	2	1	150–300
3	4	2	1	350–650	1	1	1	150–300
4	8	1	2	600–1000	3	3	1	350–650
5	4	3	1	425–750	3	4	0	350–650
6	0	2	0	100–220	0	1	0	40–150
7	5	3	1	500–825	2	1	1	200–400
8	15	6	4	1350–2250	4	3	4	600–1000
9	1	1	1	150–300	0	2	1	150–300

was calculated both directly and, assuming two 2½-inch cores to be equivalent to one 4-inch core, from Table 1 in Anon. (1944). The population per acre calculated directly fell well within the limits calculated from the table, so the latter, together with the size distribution of the wireworms concerned, is given in Table 1 above.

The numbers of wireworms longer than 5 mm were much the same both as between ridge and furrow and as between plots, except for plot 8 where there were slightly more on the ridges. The greatest differences occurred with wireworms below this size (i.e. probably the 1955 generation), of which most were recovered from the ridges, principally on plots 4 and 8. By comparison with the larger wireworms, which were all found singly except for one core which contained two, the smaller ones were aggregated, with between two and five per core. Since fifteen small wireworms were recovered from plot 8 it seems unlikely that their relative absence from the other samples was due to faulty technique, particularly since Salt and Hollick estimated that their method recovered over 90 per cent of the population. The summer of 1955 was particularly dry and either oviposition or survival of eggs may well have been restricted to the plots which withstood the drought best.

In the summer of 1955, in addition to the above samples, large cores (8 in. diameter and 12 in. deep) were taken; nine from the furrows and nine from the ridges of each plot. These were hand sorted for earthworms, partly by the present author and partly by Dr. J. A. Svendson. The wireworms encountered during sorting were preserved, and all individuals longer than 5 mm, from both large and small cores, were identified as far as possible according to the key published by Van Emden (1945) after specimens had been sent to that author for sample determination. *Agriotes lineatus* L. and *Agriotes obscurus* L. were separated by the shape of the pre-apical mandibular teeth, their identity being checked by the shape of the spiracles. Where the mandibles were very worn or where the determination was doubtful for any other reason the specimen was listed as unknown. As pointed out by Van Emden in the key, abrasion of a *lineatus* type mandible can produce an *obscurus* type but not vice versa, so that typical *lineatus* individuals must belong to that species. The species composition of the total wireworms longer than 5 mm recovered from the plots is given in Table 2.

TABLE 2

Species Composition of Total Wireworms Longer than 5 mm from Tree Field

Plot	*Agriotes lineatus* L.	*Agriotes obscurus* L.	*Corymbites cupreus* F.	*Hypnoidus riparius* F.	Unknown	Total
1	0	11	0	0	1	12
2	4	16	0	0	1	21
3	14	5	2	0	2	23
4	20	7	2	2	2	33
5	0	19	0	0	2	21
6	3	13	0	0	1	17
7	9	9	1	0	3	22
8	34	7	0	0	3	44
9	14	4	0	0	2	20

The populations were composed principally of *A. lineatus* and *A.obscurus*, the former predominating on plots 3, 4, 8 and 9 and the latter on plots 1, 2, 5 and 6, with roughly equal numbers of both species on plot 7. That is, *A. lineatus* was most abundant on four out of the five plots receiving basic slag and *A. obscurus* was most abundant on the others, except for the fifth slag plot where the two species were found in roughly equal numbers.

Some evidence that the relative abundance of *A. lineatus* and *A. obscurus* was related to percentage loss on ignition and to pH of the soil was given by data obtained by R. Brockman and B. Roscoe (King's College), who set out a fertilizer trial on plots 4 to 9 after the field was ploughed.

TABLE 3

Percentage Loss on Ignition and pH of Tree Field Plots. (Means of Six Readings per Plot before Ploughing)

	A. obscurus dominant		*A. lineatus* dominant		
	Plot 5	Plot 6	Plot 4	Plot 8	Plot 9
Per cent loss on ignition ..	8·7	7·6	9·5	10·1	8·9
pH	5·5	5·5	5·9	7·4	6·3

That is, *A. lineatus* was most abundant on plots where pH and percentage loss on ignition were high and *A. obscurus* on plots where they were low. This may be compared with the results of Salt and Hollick (1946) who found that the abundance of *Agriotes sputator* L. in one field at Cambridge was correlated, positively with the former and negatively with the latter factor. These authors also found that the abundance of *A. sputator* was negatively correlated with the amount of *Lolium* in the sward. Specific Frequency counts of the composition of the Cockle Park swards in 1950 suggested that the plots on which *A. lineatus* was most abundant, and plot 2 of the *A. obscurus* series, had more *Lolium*, *Dactylis*, *Cynosurus*, *Trifolium* and *Ranunculus* than the rest. These differences were not revealed again in this order, however, in a second percentage area analysis made in December 1955 by another observer (F. Blackburn, King's College).

Salt and Hollick, including *A. lineatus* and *A. obscurus* in one group, found that the joint distribution of the two species was negatively correlated with that of *A. sputator*. In essence, in Tree Field, the abundance of *A. lineatus* was negatively correlated with that of *A. obscurus*. The latter, dominant on the plots of low fertility and coarse vegetation, is by far the commonest wireworm in the north of England where relatively sour infertile pastures are more usually found. *A. lineatus*, dominant on the better plots, is more usually found in the south (Anon. 1944).

An estimate of the amount of damage caused by these wireworm populations was made on four drill widths of undressed seed (spring oats) which were run in, two on each side of a central 40-yard wide strip of commercial crop, across plots 4 to 9 as supplementary to the fertilizer trial. There were no signs of attack at first but after the crop had all appeared above ground, many of the plants died suddenly during a dry spell. They had been bitten through the coleoptile, and the secondary roots which were just beginning to form were not sufficiently far advanced for them to survive. Subsequently the attack appeared to cease, and there were no further signs of damage to the maturing plants when they were sampled just after tillering. The amount of damage was assessed on two occasions, firstly when dead plants were at their most noticeable (by digging five, double row, foot lengths of drill per drill-width per plot and counting bitten coleoptiles) and secondly, after the attack had ceased but before tillering was far advanced (by counting live plants per double row, yard length of drill, in the same way). On both occasions simple analysis of variance of the results showed that the plots fell into two groups within which the amounts of damage were not

significantly different; plots 6 and 9 suffered significantly ($p = 0{\cdot}01$) less damage than plots 4, 5, 7 and 8.

The drill widths were harvested separately, but owing to the great differences in fertility between plots and to a heavy frit fly attack during tillering there was no relationship between wireworm damage and subsequent yield.

I am grateful to Dr. A. Milne, A.R.C., Unit of Insect Physiology at Newcastle, for permission to use his soil washing apparatus, to Dr. F. I. Van Emden for identifying the wireworms, and to Mr. F. Blackburn and Messrs. R. Brockman and B. Roscoe for permission to quote their results.

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CUCUMBER MOSAIC VIRUS IN RASPBERRY

by B. D. HARRISON

Scottish Horticultural Research Institute, Invergowrie, by Dundee

BECAUSE the Lloyd George variety of raspberry appeared to be immune from infection by raspberry ringspot virus in grafting experiments (Cadman, *in lit.*), crops of Lloyd George have been examined rather carefully, primarily to see whether or not the variety is immune in field conditions. Although no plants infected with raspberry ringspot virus were found, a previously unknown disease was observed in two fields. A description of this disease, and the studies which led to the conclusion that it is associated with a strain of cucumber mosaic virus, is given below.

THE DISEASE IN LLOYD GEORGE RASPBERRY

Plants showing typical disease symptoms were first found in a four-year old plantation of the New Zealand stock of Lloyd George growing near Perth, Scotland. These plants had large light and dark green blotches on their leaves, but their growth seemed unaffected by infection, and the disease probably has little or no effect on fruit yield. Plants with these symptoms were scattered irregularly through the plantation in a way which suggested that some of the canes were infected when planted, but there is no proof that this was so. The low incidence of the disease suggested that the virus was not spreading rapidly. The distribution and incidence of the characteristic symptoms were similar in a second field several miles distant from the first.

Sap from blotched raspberry leaves produced a few local lesions in inoculated leaves of *Chenopodium amaranticolor*, but sap from normal-looking plants, or

from those with other kinds of symptoms, did not. Further tests confirmed that only extracts from plants with blotched leaves caused lesions in *C. amaranticolor*. The infected Lloyd George plants probably also contained one other virus, the Lloyd George latent virus (Cadman, *in lit.*), which is carried without causing symptoms. The symptoms described above therefore refer to plants infected with both the sap-transmissible virus and Lloyd George latent virus. The former virus has not been found in other raspberry varieties.

HOST RANGE AND PROPERTIES OF THE VIRUS

The sap-transmissible virus was cultured from the local lesions in *C. amaranticolor* leaves and eventually transmitted to *Petunia hybrida*. This plant was used as a source of virus for further mechanical inoculation. The symptoms produced in different species are given below:

Beta vulgaris L. (sugar beet). Chlorotic local lesions and systemic green mosaic: systemically infected leaves were abnormally small and narrow.

Chenopodium amaranticolor Coste & Reyn. Local lesions with a necrotic centre and yellow margin: not systemic.

Cucumis sativus L. (cucumber). Chlorotic local lesions and systemic yellow mosaic.

Datura stramonium L. (thorn-apple). Faint chlorotic local lesions and systemic green mosaic.

Lycopersicon esculentum Mill. (tomato). The laminae of systemically infected leaves were narrow, but not extremely so.

Nicotiana tabacum L. var. White Burley (tobacco). Faint chlorotic local lesions and systemic green mosaic: the systemically infected leaves formed later were almost symptomless.

Petunia hybrida Vilm. Faint chlorotic local lesions, and vein-clearing on the first of the systemically infected leaves: systemically infected leaves produced subsequently had an indistinct green mosaic.

Phaseolus vulgaris L. var. Prince (French bean). In winter very small brown lesions were formed in the inoculated leaves, but not in summer: not systemic.

In vitro tests. Infectivity was destroyed by heating infective tobacco sap for 10 minutes at 68°C but not at 62°C. The dilution endpoint of the virus in tobacco sap was between 1: 1000 and 1: 10000 when "Celite" was mixed in the inoculum.

PLANT PROTECTION TESTS

Preliminary tests in *P. hybrida* and tobacco showed that the virus from Lloyd George was not a strain of raspberry ringspot virus or potato virus Y, and suggested that it was related to the yellow strain of cucumber mosaic virus (YCMV). Further tests in *P. hybrida* and tobacco, in which YCMV normally produces systemic symptoms within a week, showed that these symptoms took 3–4 weeks to appear in plants previously infected with the Lloyd George virus. In tobacco, a few yellow local lesions sometimes developed in inoculated leaves, whereas YCMV caused very numerous lesions in leaves of healthy plants. Similarly, when YCMV-infected *P. hybrida* plants were inoculated with the Lloyd George virus, the slight change in type of systemic symptom did not occur until six weeks after inoculation. Thus, although these results suggested that the Lloyd George virus might be related to YCMV they were not conclusive. However, tobacco plants infected with the Lloyd George virus were immune from infection by a strain of cucumber mosaic virus from spinach which causes local and systemic necrosis in tobacco (Bhargava, 1951). The Lloyd George virus therefore seems to be a strain of cucumber mosaic virus, closely related to the spinach strain and more distantly related to YCMV.

TRANSMISSION BY APHIDS

Green peach aphids (*Myzus persicae* Sulz.) transmitted the virus quite readily from *P. hybrida*, cucumber or sugar beet to *D. stramonium* or tobacco when given an acquisition feed of five minutes and test feeding period on the healthy plants of twenty-four hours. *M. persicae* has not, however, been found on raspberry in the field, and is unlikely to be a natural vector in raspberry plantations. The ability of the large raspberry aphid (*Amphorophora rubi* Kltb.) to transmit the virus from *D. stramonium* to black raspberry (*Rubus occidentalis* L.) was, therefore, tested together with that of *M. persicae* and *Macrosiphum euphorbiae* (Thos.). *R. occidentalis* was used in preference to red raspberry, because it is much more susceptible than the latter to infection with other raspberry viruses transmitted by *A. rubi*. The acquisition feed was 2–5 minutes and the test feeding period was twenty-four hours. All three aphid species made short feeds on both infected and healthy plants, but *M. euphorbiae* fed best on *D. stramonium*, and *A. rubi* fed best on *R. occidentalis*. Twenty-four *R. occidentalis* seedlings, five aphids per seedling, were used for each aphid species. Only two plants which had been infested with *A. rubi* developed symptoms, a green mosaic on a few leaves. The virus was isolated from one of these two plants by mechanical inoculation, and later proved to be still transmissible by *M. persicae* from cucumber to tobacco. No local lesions were produced in *C. amaranticolor* leaves by sap from thirty-six of the symptomless *R. occidentalis* plants: twelve plants were tested from each of the lots previously infested with the different aphid species.

Several attempts were made to transmit the virus from raspberry to *D. stramonium* using *A. rubi* and *M. persicae* but without success.

CONCLUSIONS

The behaviour of this virus from Lloyd George raspberry, in plant-protection tests, shows that it is a strain of cucumber mosaic virus. The symptoms produced in test plants, thermal inactivation point and aphid transmissibility all support this conclusion. Although cucumber mosaic virus has been found in plants of many species and has been transmitted by several aphid species (Smith, 1957), raspberry seems to be an unrecorded host and *A. rubi* an unrecorded vector.

It seems quite possible that cucumber mosaic virus is never, or only rarely, transmitted from raspberry to raspberry in the field but that, having been once transmitted from some other species to raspberry, its incidence in the latter depends on the propagation of infected plants.

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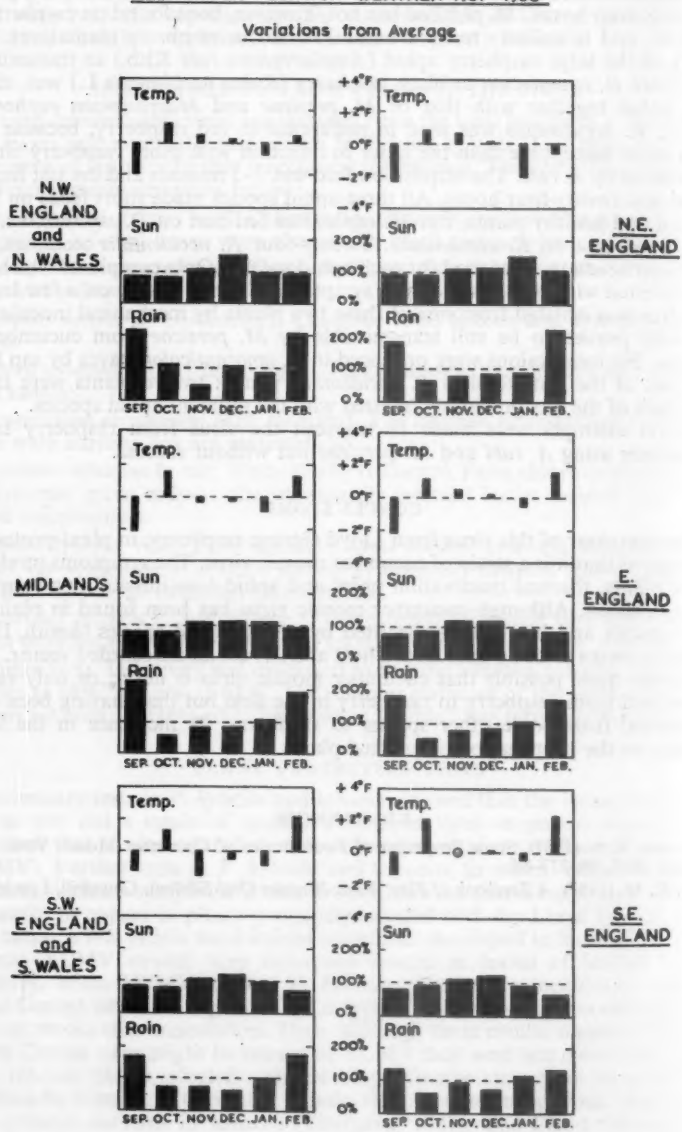
SEPTEMBER–FEBRUARY WEATHER 1957–1958
Variations from Average
N.W. ENGLAND and N. WALES
N.E. ENGLAND
MIDLANDS
E. ENGLAND
S.W. ENGLAND and S. WALES
S.E. ENGLAND
Temp.
Sun
Rain
+4°F
+2°F
0°F
−2°F
−4°F
200%
100%
0%
SEP. OCT. NOV. DEC. JAN. FEB.

THE WEATHER OF ENGLAND AND WALES*

AUTUMN AND WINTER 1957–58

As is often the case, September followed the example of August in its weather behaviour; it was a cool month, with stormy periods and below average sunshine; the rainfall was well above average in all areas except south-east England where more normal conditions prevailed. October and November brought quieter and drier conditions; the first half of October, and all except the first week of November, was unusually settled in character, although heavy rain during the fortnight common to both months tended to obscure this fact in the monthly totals. This late autumn period was generally mild with occasional bursts of colder weather. The first frosts were later than usual.

The winter opened with changeable but generally mild weather, being slightly drier than normal in the south of Britain. Similar weather prevailed in January until the 18th, when there followed a week of cold northerly winds bringing unusually severe night frosts with snow in most parts, especially in the northern areas. February had three similar cold spells of short duration but intervening days were generally mild and wet. Snowfall was heavy at times in the north but was generally followed by thaw conditions after two to four days. The total precipitation for the month (rain and snow) was generally high.

The weather for each of the six months, September 1957 to February 1958, is represented graphically on page 112. Figures for each of the six districts of England and Wales are shown diagrammatically in terms of deviation from average of air temperature, sunshine and rainfall. The wet conditions of September and February are outstanding in comparison with the drier weather of the other four months. The inclement September was compensated for by a more "open" condition in late autumn and, despite February snowfalls, January was in general the coldest month in a winter which was not a conspicuously cold one.

NEW OR UNCOMMON PLANT DISEASES AND PESTS

Perfect state of *Phoma betae* found in England. In October 1957, the perithecia of *Pleospora betae* Björling were found on the dried stems of wild beet, *Beta vulgaris*, at Dunholme, Lincolnshire (Plate II, 3). The stems, which came from an experimental plot, were stacked in the open in July and kept under observation. The minute, black, circular fructifications were first noticed in September. They were best developed on the upper illuminated sides of the thicker part of the stems, but were also present on the flower stalks, bracts and seed-balls. Sectioning showed that they were a mixture of perithecia and pycnidia of *Phoma betae* Frank. The perithecia were common from October 1957 to March 1958. Their

* These notes and the diagram on page 112 are published by permission of the Director-General of the Meteorological Office.

presence was demonstrated by placing a slice of stem bearing the fructifications face downwards on a microscope slide, and placing the whole in damp air. In the course of one or two days the muriform, tinted ascospores were ejected on to the glass. Similar but fewer perithecia were also found in January 1958 on sugar-beet seed crop stems, which had been cut in August, dried on tripods, and stacked in the open in October. Cultures on potato dextrose agar showed that the organism derived from an ascospore was identical with that obtained from a pycnospore of *Phoma betae*, thus proving that the ascogenous and conidial forms belonged to the same fungus. The only previous record of perithecia of *Pleospora betae* is from Sweden, where they were found on buried stems of sugar-beet (K. Björling, *Bot. Notiser*, 1944, 215–22). Specimens have been deposited at the Commonwealth Mycological Institute, Ferry Lane, Kew, Surrey. Thanks are due to Mr. E. W. Mason and Mr. C. Booth of this institute for confirming the identification of the fungus and for cultural work, and to Mr. V. Stansfield for the photograph.

C. E. CORNFORD

A Nematode Genus new to Europe. A sample of galled tomato roots from a nursery at Wokingham, Berks, was submitted to the advisory entomologist at Reading in September 1957. The galls were thought to be caused by root-knot eelworm, but they were small in comparison with the usual galls, and in most cases were rather flat with a rough, corky surface (Plate III, 2). The material was examined by Dr. M. T. Franklin of Rothamsted, who found that the galls contained a species of *Nacobbus*. This genus has, up to the present, been recorded only from Utah, California, Nebraska, Colorado and Wyoming, where three species are known. The present species does not agree fully with the descriptions of any of them: a mature female is shown in Plate III, 1. The infested plants had been grown in a heated glasshouse in loam bought from Surrey, reputedly from a source used by the nursery for several years. Galling of tomato roots has been noticed throughout this period, and the grower assumed that root-knot eelworm was the cause. Soil treatment with parathion had been carried out regularly, and no serious loss of crop was noticed. It is possible that *Nacobbus* sp. has been present on the nursery for some years, but nothing can be said as yet about its pest status. A range of likely host plants is being grown in *Nacobbus*-infested soil at Rothamsted and Reading. The photographs were taken at Rothamsted, Plate III, 1 by Mr. C. C. Doncaster, and Plate III, 2 by Mr. V. Stansfield.

C. W. GRAHAM

New host records for root-knot eelworms. *Meloidogyne incognita* (Kofoid and White) Chitwood, possibly mixed with the variety *acrita* Chitwood (det. M. T. Franklin) were found in the galls of pot-grown plants of *Cyclamen persicum* Mill. with heavily galled roots which were received from a nursery in Middlesex in November 1957. Over 1,000 plants were affected. B. D. MORETON

Meloidogyne incognita var. *acrita* has been identified from root galls of *Hoya* sp. received in 1952 from a Surrey glasshouse. Another root-knot eelworm, *M. hapla* Chitwood, was found in root galls on the following plants from two nurseries in East Suffolk: several *Clematis* hybrids grafted on *C. vitalba* L. in 1956, both stock and scion roots being infested; and *Clematis* Mme Edouarde Andre in 1957 from the first nursery; *Antennaria dioica* (L.) Gaertn. in 1956, and *Diervilla* x *styriaca* Hort. in 1957 from the second nursery. The *Antennaria* were pot-grown in cold frames from divisions of outdoor parent plants and the *Diervilla* was grown out of doors. J. F. SOUTHEY